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PAINFUL PHANTOM LIMB TREATED BY HIGH CERVICAL CHORDOTOMY

REPORT OF TWO CASES

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THE lot of a patient, who after amputation experiences pain in his missing limb, may become pitiful. Indeed, some of these sufferers have sought release by suicide. Although it is well known that an absent limb commonly persists as a phantom, it is not generally appreciated that it may become the seat of genuine pain, and all too often the unhappy patient is regarded as the victim of a psychological disturbance.

Judging by reports in the literature, the treatment of painful phantom limbs has been for most surgeons a disappointing experience (Bailey and Moersch, 1941; White, 1944). In a few cases the excision of tender end-bulbs on the divided nerves or adherent scars in the stump has proved beneficial. In others interruption of the sympathetic pathways from the limb, either by infiltration with a local anæsthetic or by sympathectomy, has relieved pain. Sometimes, indeed, a single injection of local anæsthetic around the sympathetic ganglionated chain has produced prolonged and even permanent benefit (Leriche, 1939; Livingston, 1938). Unfortunately, however, these simple measures do not relieve all patients, and when they fail the problems of treatment become difficult and formidable. Many procedures have then been tried, but none have gained general acceptance. Destructive procedures on peripheral nerves, nerve-plexuses, or nerve-roots, and the intra-arachnoid injection of alcohol, have usually failed (Riddoch, 1941; White, 1944). Division of the lateral spino-thalamic tract within the spinal cord has proved successful in some hands for painful phantom in the lower limb (White, 1944), but not in all (Bailey and Moersch, 1941); and recently the operative excision of areas of the sensory cortex has been recommended instead (de Gutiérrez-Mahoney, 1944). In fact surgical treatment has failed so often that many authorities regard patients with painful phantom limb as psychoneurotic (Bailey and Moersch, 1941), and some surgeons have even considered performing prefrontal leucotomy (White, 1944), a procedure which is only doubtfully justifiable.

One of the difficulties with spinothalamic tract section has been to secure a sufficiently high level of analgesia, particularly when the upper limb is concerned. To be effective the tractotomy must produce complete analgesia not only of the segmental levels of the missing limb, but also for a short distance above this. If the lower limb alone is concerned, the necessary analgesia can readily be obtained by anterolateral chordotomy performed in the upper thoracic region. If, however, the upper limb is involved, it is difficult to produce the necessary extent of analgesia by tract section no matter how high the level at which it is performed. This may explain why, when high cervical chordotomy has been tried for painful phantoms in the upper extremity, only partial successes have been observed (Foerster and Gagel, 1932; de Gutiérrez-Mahoney, 1944; White and Smithwick, 1942). J. C. White (1944) with his great experience states, "So far I have had no opportunity to attempt a high section of the spinothalamic tract for phantom pain in the arm, and have been unable to find any successful report of its accomplishment".*

It seems appropriate, therefore, to describe 2 cases of painful phantom in the upper limb which were relieved by high cervical chordotomy performed by controlled graduated division under local anæsthesia. Neither had been benefited by previous blocking of the sympathetic and peripheral nerve pathways. Observations were made in them which throw light on the mechanism of pain-production in painful phantom limbs. Together they show that at least some cases of painful phantom upper limb, hitherto regarded as intractable, can be relieved by operation.

CASE REPORT

Case 1.—**Army corporal, aged 27 years, suffering from causalgia in a phantom hand following traumatic amputation of an upper limb.**

* Since forwarding this paper for publication, we have learnt that Mr. Lancelot Bromley has an apparently successful case, which he operated on in 1923 and published in *Guy's Hospital Reports* for April, 1930. The anterolateral column on the contralateral side of the spinal cord was divided at the level of the 3rd cervical segment, while the 3rd and 4th posterior cervical roots were divided on the same side. The patient was completely relieved of all pain in his phantom upper limb. When examined four years later he exhibited impairment of pain sensibility below the 6th thoracic segment, but not above this level.

This soldier, referred in May, 1944, to the Neurosurgical Unit, Dunedin Hospital, by Lt.-Col. J. J. Brownlee, N.Z.M.C., had had his right forearm blown off at the elbow-joint by an 88-mm. shell twenty months earlier, while he was on active service in the Middle East. In addition, he sustained several superficial injuries in the base of the neck, the lobe of the right ear, and the

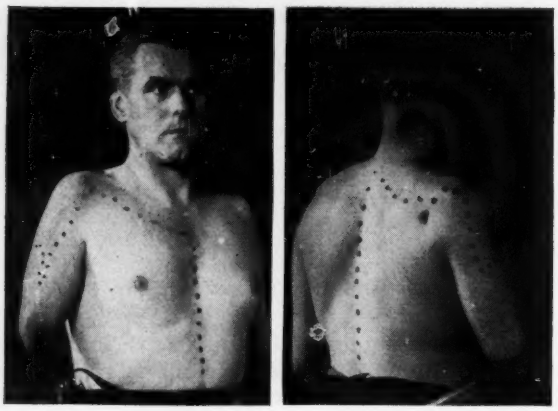

FIG. 343.—*Case* 1. Photograph after operation, showing appearances of stump as well as the upper limits of area of analgesia.

right temporal region from fragments of the exploding shell, but these did not cause residual symptoms. He was unconscious for a minute or two. Four hours later at a C.C.S. his arm was re-amputated through uninjured tissues at the junction of the middle and lower thirds of the humerus. The stump healed promptly without any trace of infection.

From the time of wounding this soldier had complained of a severe, constant burning pain in his phantom right hand. It was most evident in the thumb and index fingers, but from these digits it spread across the knuckles to involve the middle and little fingers, and also radiated to the dorsum of the wrist. Usually the pain was bearable, but at times it became intense. The phantom hand, itself, felt of normal size and shape, except that the ring finger was completely missing. It was connected by a shortened phantom forearm to a phantom elbow perched at the end of his stump. The phantom forearm and hand were held flexed, as in a sling. The phantom fingers could be moved voluntarily, but with great mental effort; otherwise the limb was immobile. At times the fingers would slowly extend spontaneously, and these involuntary movements were associated with agonizing intensification of his pain. Otherwise there were no known aggravating factors, and in point of interest neither morphine nor alcohol appeared to have much influence on his appreciation of pain. He had undergone three secondary operations on his stump for the excision of terminal neuromas and for alcohol infiltration of the major nerve-trunks, all without relief.

This man's files showed he was a brave soldier. He was a well-built, intelligent fellow, who had been regarded with suspicion by psychiatrists "particularly because his pain did not appear to have an anatomical distribution". He had been examined several times under pentothal narcosis, but nothing was found in his past record or in his family history to suggest an inborn tendency to develop undue psychological strain. We were therefore surprised to find on examination nothing to explain his symptoms. His amputation stump was faultlessly constructed, and of normal colour and temperature (*Fig.* 343). At least two small neuromas could be palpated well away from the end of the bone, but though these produced tingling sensations in the phantom when pressed upon, they did not affect his spontaneous pain.

Here, then, was a man of exemplary type, complaining bitterly of pain in a phantom limb, but without any features either in his stump or elsewhere to account for it. We decided to test out the effect of blocking of the sympathetic and peripheral nerve pathways.

The upper three thoracic sympathetic ganglia on the right side were infiltrated each with 10 c.c. of 1 per cent procaine (modified Labat's technique). The right pupil constricted (Horner's syndrome). The skin temperature of the stump recorded by a thermocouple rose gradually within a quarter of an hour from 26° to 32° C., whereas simultaneously the temperature of the corresponding point on the sound limb fell from 26° to 24° C. (room temperature 21° C.). Although a sympathetic block was thus shown to be present, the pain remained unabated. He, however, volunteered that his phantom hand felt warmer than before the test.

At the end of an hour, and while the sympathetic block was still complete, the brachial plexus in the posterior triangle of the neck was infiltrated with 40 c.c. of 1 per cent procaine. This resulted in complete analgesia and anæsthesia of the stump, including both the anterior and the posterior axillary folds, and also the outer aspect of the arm up to the insertion of the deltoid muscle. The patient said that his pain was still unaltered, although his hand now felt colder. Thus the pain had not been relieved by the measures usually advocated. This failure of response to blocking of the brachial plexus indicated that any further operative attacks on peripheral nerves, and probably on posterior nerve-roots as well, would be fruitless. The man was desperately anxious to be relieved of his pain, and prepared to go to any reasonable lengths. Operative section of the spinothalamic tract was therefore advised.

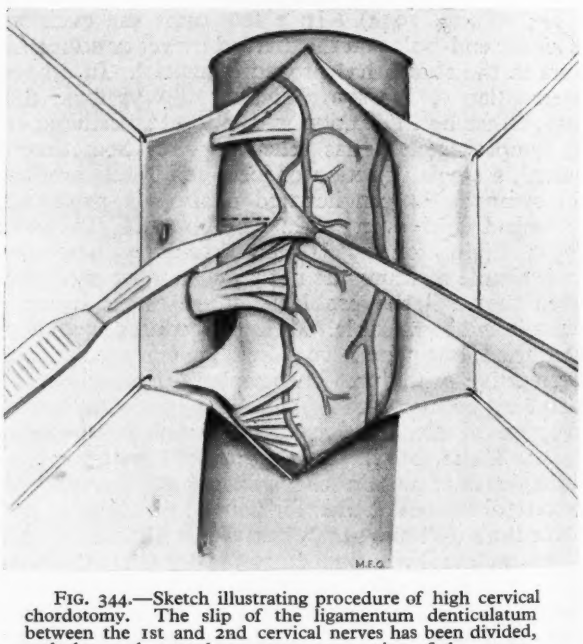

FIG. 344.—Sketch illustrating procedure of high cervical chordotomy. The slip of the ligamentum denticulatum between the 1st and 2nd cervical nerves has been divided, and the cord rotated preparatory to section of the anterolateral column.

Laminectomy of the upper three cervical vertebræ was performed under local anæsthesia after preliminary sedation with morphine (gr. ¼) and with the patient prone. The spinal cord appeared normal. The slip of the ligamentum denticulatum on the left side between the 1st and 2nd cervical nerve-roots was divided, and the cord rotated through about 45° in order to bring the left anterolateral column into view (*Fig.* 344). Next, a tenotome was inserted to a depth of 2·5 mm. midway

between the 1st and 2nd cervical roots and about 2 mm. in front of the line of attachment of the ligamentum denticulatum, and was then carried forwards until its point emerged on the surface in the line of the anterior nerve-roots. This cut was painless, but we noted that the patient complained of headache whenever arachnoidal strands spanning the subarachnoid space were pulled on, whenever the cord was rotated sufficiently to put nerve-roots on the stretch, or whenever sufficient cerebro-spinal fluid was sucked away to dry the cord within the foramen magnum.

This first cut had no effect on the painful phantom, but the anæsthetist reported that it had numbed the body below the anterior axillary fold. It was therefore deepened to between 3 and 4 mm., whereon the patient said that his pain was now vague, and examination disclosed that the inner side of the stump was numbed to pin-prick but not the outer side. A third cut was then made to just over 4 mm. deep and was carried forwards to between 3 and 4 mm. in front of the anterior nerve-roots (*Fig.* 345). The patient now volunteered his pain was gone. At no stage of the operation was the patient

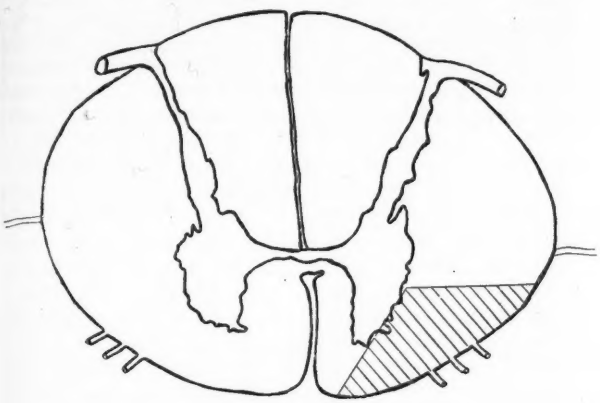

FIG. 345.—Diagrammatic cross-section of the cervical cord to show extent of the chordotomy in *Case* 1.

told of what was being done, or of what sensations he could expect. His behaviour impressed all by its genuineness. During the closure of the laminectomy wound, general anæsthesia was induced with sodium pentothal administered intravenously. The patient's convalescence was uneventful.

Following operation the patient continued to be aware of his phantom, but no longer felt pain in it. He exhibited certain interesting neurological signs. These were on the right side complete loss of pain sensibility, of temperature sensibility, and of tickle sense over the limbs and trunk below the 4th cervical dermatome (*Figs.* 343, 346), and on the left side a Horner's syndrome evidenced by ptosis of the left upper eyelid, by constriction of the left pupil, by gross diminution of thermo-regulatory sweating (Guttman's chinazarin method) over the left side of the face, head, and upper limb, with only slight impairment over the left side of the body below that, and by diminution of the pilomotor reflex on the left side of the chest. These left-sided signs suggest that the left tecto-spinal tracts had been divided in addition to the spinothalamic tract. There was no detectable impairment of tactile, postural, vibrational, and 2-point discriminative sensibility on either side of the body, except for a narrow zone of diminished sensibility over the upper part of neck on the left side (? C.2 dermatome). There was no loss of testicular pain on either side.

This patient was discharged from hospital 3 weeks after operation. When seen more than a year later, his phantom was still painless and his residual sensory neurological signs were unchanged. The left-sided Horner's syndrome had disappeared. He was back at work without any disability, other than that due to the absence of the amputated limb.

Case 2.—**War pensioner, aged 33 years, suffering from casualgia in a phantom hand following surgical amputation of an upper limb.**

This man, referred by Dr. R. H. Q. Baxter, had had his left elbow shattered by a sniper's bullet while serving in Crete in May, 1941. A field dressing was applied, and then under great difficulties he was evacuated to Egypt. By the time he reached Alexandria he had

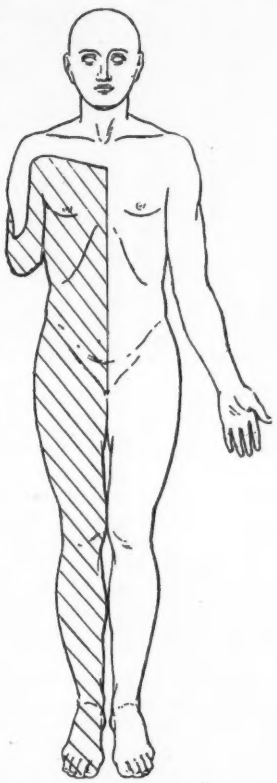

FIG. 346.—*Case* 1. Extent of loss of pain, temperature, and tickle sensibility following chordotomy. Touch and postural sensibility were preserved.

developed gas gangrene, and so 8 days after wounding his arm was amputated below the upper third of the humerus. He cannot recall the events of the next month, probably on account of toxæmia. His stump became septic, and more than five months went by before it healed.

As soon as he regained consciousness he was aware of pain in the phantom hand and in his stump. The phantom hand felt a little smaller than the normal, and lay at some distance from the stump. It was, moreover, incomplete, for the only portions which he could definitely discern and in which he could locate his pain were the thumb, which he could identify separately, and the fingers, which he could only group together. These digits were held semiflexed, and kept squirming together continuously. Voluntarily he could move them a little, but the effort made his pain intolerable. The pain also became severe with emotional stress, such as seeing blood-curdling pictures at the cinema or hearing other people discuss their wounds. It was not affected by pleasurable excitement, nor by morphine or alcohol. In addition to the pain in the phantom, there was also a continuous but less severe

ache throughout the stump. A year after wounding he underwent an operation for trimming the stump and excising neuromas, but without relief.

This man's past record and bearing, like that of *Case* 1, were beyond reproach. On his return to New Zealand towards the end of 1941 he had re-entered the Civil Service as a clerical worker, and in spite of his pain had risen to a responsible post. It was in the course of his official duties that he encountered the first patient, and, impressed by the result, he initiated arrangements for his own admission to the Neurosurgical Unit.

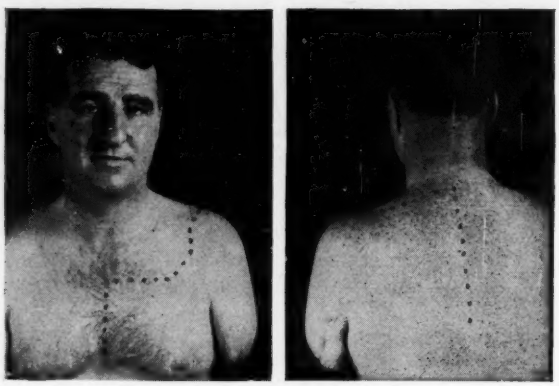

FIG. 347.—*Case* 2. Photograph after operation, showing appearances of stump as well as upper limits of area of analgesia. The drooping of the right upper eyelid, the constriction of the right pupil, and the laminectomy incision can also be seen.

On examination little abnormal was found. The stump was well-formed, and of normal colour and temperature (*Fig.* 347). Pin-prick and light touch were felt a little more acutely than usual over the end of the stump, but there was no tenderness. Several neuromas could be palpated, and pressure over some of them caused pain in the fingers. The same investigations were made as in *Case* 1. A procaine block of the upper three thoracic sympathetic ganglia had no effect on the pain, although the block was complete as demonstrated by the occurrence of a Horner's syndrome and by the complete absence of thermo-regulatory sweating in the left face and left fore-quarter. Similarly anæsthetization of the brachial plexus with procaine solution was without benefit.

This man, then, was a case of painful phantom limb similar in most respects to the first. The only points of difference were that the initial amputation had been surgically performed and not traumatic, while his wound had been infected. Although we cannot tell whether pain was present in his phantom immediately following amputation, it was noticed immediately clear consciousness was regained some four weeks later. These differences, we felt, were not fundamental. The same procedure as in *Case* 1, namely, a high cervical chordotomy, was therefore recommended.

The result was similarly beneficial, but incidently revealed an important hazard in this operation. Laminectomy of the upper three cervical vertebræ was performed under local anæsthesia, and the cervical cord exposed by opening the dura. Rotation of the cord proved slightly more difficult than in *Case* 1, and before it could be accomplished properly the upper three slips of the ligamentum denticulatum on the right side had to be divided. It is interesting that rotation of the cord and traction on subarachnoidal strands again caused pain, although sucking out of cerebrospinal fluid this time had no effect. The anterolateral column was then sectioned in stages at the same level as in *Case* 1 (i.e., midway between C.1 and C.2 nerve-roots). Before this could be done a tiny artery on the surface of the cord which crossed the line of section had to be coagulated with a finely-controlled diathermy current applied via a fine hook, and then divided. The first cut to a depth of 2·5 mm. produced analgesia extending up to the 3rd thoracic segment only, and did not affect the phantom. As the cut was deepened the patient said his spontaneous pain lessened, but it did not finally disappear until the point of the tenotome had penetrated the cord to a depth of between 4 and 5 mm. immediately in front of the line of the ligamentum denticulatum, and had emerged on the anterior surface 3 to 4 mm. in front of the line of the anterior nerve-roots. The cuts were bloodless. The patient now volunteered that he was free of pain, but to our consternation his right arm and right leg appeared paralysed, although just prior to the last cut he had moved them well. The closure was then carried out under intravenous pentothal anæsthesia.

Fortunately within an hour of returning to the ward he once more began to move his right limbs. Thereafter his convalescence was straightforward. He proved to have been completely relieved of his pain, but his phantom persisted. He showed similar post-operative signs to *Case* 1, viz. loss of superficial and deep pain sensibility and of temperature sensibility on the left side below the C.3 dermatome, and a right-sided Horner's syndrome associated with diminished thermo-regulatory sweating over the right side of the body, marked in the upper half and only slight below the costal margin (*Figs.* 347, 348).

FIG. 348.—*Case* 2. Extent of loss of pain, temperature, and tickle sensibility following chordotomy. Touch and postural sensibility were preserved.

Once again the sensations of touch, posture, vibration, and 2-point discrimination were unaffected. Fortunately he had no residual weakness of the right limbs, although the right abdominals were diminished, and the right plantar response remained extensor for three months before becoming normal again.

This man was discharged from hospital three weeks after operation and shortly afterwards resumed work. When re-examined six months later he was still free of

pain in his phantom hand, which in the meantime had receded until it projected from the end of his stump. The Horner's syndrome had disappeared, but the other neurological signs remained. Although he no longer had pain and temperature sensibility below the left clavicle, he remarked that, if he wore rough clothes or had a hot shower bath, he felt an annoying sense of pins and needles in his trunk and left thigh. These sensations were only occasionally present and did not inconvenience him. His only disability was the loss of his limb.

DISCUSSION

The descriptions of their painful phantoms given by our two patients are similar to accounts of other cases related by Riddoch in his presidential address on "Phantom Limbs and Body Shape," delivered to the Neurological Section of the Royal Society of Medicine in 1941. Fortunately the medical profession and the educated laity are now sufficiently well informed to regard no longer such accounts with disbelief or even with accusations of insanity. The purely descriptive details, therefore, need not be further considered. The more important features are those which throw light on the mechanism of pain production in phantom limbs and their treatment.

Probably much of our confusion of thought regarding painful phantoms arises from failure to recognize that there is more than one cause of pain. Many cases are relieved by interruption of peripheral nerve or sympathetic nerve pathways, but a few, as exemplified by our own two patients, are not. Surely this suggests that in these two groups the mechanisms by which pain is produced are different. It may be profitable, then, to inquire whether there are any other differences between these two groups so that we can learn to recognize and distinguish them more readily, and hence to treat them with greater understanding.

Leriche (1939) points out that usually pain does not appear in a phantom limb until some weeks or even months have elapsed since amputation, and moreover is often associated with burning sensations and with vasomotor and trophic disturbances in the stump itself. The exciting factor appears to be one of irritation of nerve-fibrils either within or around the neuromas which form at the ends of divided nerves. Leriche states that when the nerve-fibrils primarily excited are peripheral nerve-fibrils within the neuroma, pain can be relieved by blocking on sections of the nerve-trunk; when, however, the nerve-fibrils primarily excited are sympathetic nerve-fibrils surrounding the neuroma, pain can be relieved by sympathetic block or sympathectomy. Such a subdivision does not concern us here, for the type of case which we have described is that in which procedures on peripheral and sympathetic nerves prove unavailing, and in which pain appears to arise in nerve-cell stations situated within the central nervous system.

Leriche also describes a small group in which pain in an absent limb appears immediately following amputation, is not associated with any appreciable disturbances in the stump, and is not relieved by infiltration of the appropriate sympathetic and peripheral nerves with local anæsthetic solutions. Our two cases probably belong to this small group, although in only one could we be certain that pain appeared immediately on amputation. The mechanism of pain production in this group Leriche regards as "inexplicable", and he likens it to "a sort of instantaneous fixation in the memory of a scene suddenly realized and converted into an obsession". In our two cases, however, we have strong evidence that symptoms were not hysterical, for in both during the course of operative procedures the nature of which the patients were unaware of, pain was lost at the appropriate moment.

By infiltrating the sympathetic and/or peripheral nerve pathways with local anæsthetic solutions, we can gain information which will guide us in treatment. Sympathetic block may prove of therapeutic as well as diagnostic value, because relief, even when conferred by only a single infiltration, may be prolonged or even permanent (Leriche, 1939; Livingston, 1938). If, then, there should be a recurrence, the sympathetic block can be repeated or else a sympathectomy performed. Similarly, when infiltration of the peripheral nerve-trunks affords relief, excision of neuromas can be considered. In this connexion Leriche raises a word of caution, for he points out that neuromas will recur, as their formation is the usual repair mechanism after nerve severance, and probably will again cause trouble. He therefore advocates that, instead of excision, the nerve-trunk above the neuroma should be divided and then resutured. Boldrey (1943), however, prefers to excise the neuroma, and to bury the stump of the nerve in a drill hole made through a neighbouring bone.

If blocking of the peripheral and sympathetic nerve pathways fails to relieve pain, destructive operations on these pathways will prove useless. Even after such procedures as the extensive division of the posterior nerve-roots and the intra-arachnoid injection of alcohol, the painful phantom persists unchanged, no matter how much the stump may be denervated (Riddoch, 1941; White, 1944). Only two types of operative procedure appear to offer prospects of relief. They are, first, division of the lateral spinothalamic tract, and, secondly, ablation of the appropriate area of the sensory cortex.

Our two cases show that, provided due technical precautions are taken, similarly beneficial results can be achieved by high cervical chordotomy for painful phantoms in the upper limb as have already been achieved by high thoracic chordotomy for painful phantoms in the lower limb. The past failures of high cervical chordotomy are responsible for the suggestion (White, 1944) that division of the spinothalamic tract within the medulla (Schwartz and O'Leary, 1941 and 1942; White, 1941) or even higher still in the midbrain (Walker, 1942) would prove after a trial to be preferable procedures. However, in none of the published reports of these operations does a sufficiently high, and yet complete, level of analgesia appear to have been obtained.

May we digress for a moment to point out how greatly the pain of a phantom limb resembles that which may occur in an intact limb with a peripheral nerve injury and which is commonly described as causalgia. The pain of both conditions has the same physical characteristics, and just as cases of painful phantom can be divided into two main groups, so also can cases of causalgia. There is first a group in which pain appears to arise in or around the

neuroma with nerve anastomosis or by sympathetic block or interruption. There is also a group which cannot be relieved by such procedures, but which we have found can be relieved by chordotomy. Indeed, for the type of pain which we are particularly considering it does not matter whether the limb is present or not.

The operation of spinothalamic tract section does not destroy the patient's perception of a phantom or of his real limb, but only abstracts from it the sensation of pain. The stump continues to be serviceable for such purposes as bearing harness or carrying an artificial limb. The fact that in this minority of cases spinothalamic tractotomy relieves pain, whereas posterior rhizotomy does not, suggests that the pain-exciting impulses arise in the nerve-cells of the posterior horn of the spinal grey matter, and pass thence via the thalamus to the cerebral cortex, where they are perceived. Possibly these nerve-cells become excitable because they no longer receive impulses from the axons of the receptor neurones in the spinal ganglia. A somewhat analogous phenomenon of spontaneous excitability occurs when nerve-cells in the sympathetic ganglia are cut off from higher control by preganglionic sympathectomy (Govaerts, 1939). Should this hypothesis be true, one might explain the observation of Bailey and Moersch (1941) that digital pressure over the main nerve-trunk above the amputation may cause the phantom to disappear, whereas cutting the nerve will not. Pressure over intact receptor nerve-fibres cut off from normal stimulation by sensory nerve-endings may excite impulses in them which pass up and temporarily control the dorsal horn cells.

So far the deliberate treatment of painful phantom limb by ablation of the appropriate sensory area in the post-Rolandic convolution has apparently been tried in only a single case (de Gutiérrez-Mahoney, 1944), although others have considered it. A successful result was obtained, but full details of the case are not given. This type of procedure is based on a clinical observation by Head and Holmes (1911) that a lesion of the parietal lobe, by destroying the recognition of posture, abolishes at the same time the phantom limb. Presumably the impulses which give rise to a phantom reach consciousness in the parietal cortex. This type of operative procedure, when compared to spinothalamic tractotomy, has the disadvantage that it destroys postural and other discriminative sensations in the stump, and so renders the stump functionally useless for such purposes as carrying an artificial limb.

Spinothalamic tract section may thus prove preferable to regional ablation of the sensory cortex. The operation of high cervical chordotomy, however, should not be entered upon lightly, for it can be difficult and laborious, especially in thick-set individuals. It is, moreover, hazardous, as the sudden onset of a hemiplegia in our second case well illustrates. This hemiplegia disappeared rapidly, indicating that the pyramidal tract had not been sectioned, and we do not know how it was produced. Penfield (1943) has described a more serious hazard, for following this operation under local anæsthesia one of his patients suddenly and inexplicably died.

Nevertheless, when a patient is suffering from genuine and intolerable pain, the surgeon must be prepared to take reasonable risks. There is sufficient evidence to justify further trials of high cervical chordotomy.

SUMMARY AND CONCLUSIONS

Pain occurring in a phantom limb has the same characteristics as causalgia occurring in an intact limb with a peripheral nerve injury. There are two main groups of cases—a group in which pain impulses appear to arise in or around the neuromas on the divided nerves and can be relieved by excision of the neuromas or by sympathetic block or interruption; and a group in which painful impulses arise in the dorsal horn cells of the spinal cord and can be relieved only by section of the spinothalamic tract or by excision of the sensory cortex of the cerebrum. Infiltration of the appropriate peripheral or sympathetic nerve pathways with a local anæsthetic solution is a useful diagnostic procedure for distinguishing clinically between these two groups. When these diagnostic tests fail to give relief, section of the spinothalamic tract is indicated and seems preferable to ablation of the sensory cortex, because it does not destroy the phantom or the patient's appreciation of his stump, but only abstracts from it the sensation of pain.

Two cases are described which well illustrate these points.

Our thanks are due to Professors W. E. Adams, F. G. Bell, J. C. Eccles, and F. H. Smirk for their help and advice; also to Miss M. E. Ogilvie and Mr. C. Weedon for assistance with the illustrations.

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VASCULAR TUMOURS OF THE SPINAL CORD ASSOCIATED WITH SKIN HÆMANGIOMATA

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ALTHOUGH a number of cases illustrating the association of vascular tumours of the brain with a nævoid condition of the face, known as Sturge's disease, have been described (Nussey and Miller, 1939), and the list is constantly being added to, the analogous combination of a vascular tumour of the spinal cord and a hæmangioma of a corresponding segment of the skin does not appear to have been so commonly observed.

Berenbruch (1890) described a case with three huge congenital angiolipomata over the right pectoral muscles in a boy aged 16; these were situated over the right scapula and latissimus dorsi muscle; there was a large plexus of veins along the spinal column connecting outwardly with an extradural angioma, and with the tumours of the back through the intervertebral foramina. There was an angioma of the cord substance itself from C.5 to D.3.

Cobb (1915) described a case of a boy, aged 4, with congenital nævi, the largest being 5 in. in diameter; there were three on the right and one on the left side, in the cutaneous areas of the 7th, 8th, and 9th dorsal segments. A cluster of large pulsating vessels was found on operation, extending from the 4th to 9th dorsal segments of the spinal cord. Cobb therefore suggested that nævi are of diagnostic value when a spinal cord lesion is present and that vascular tumours might be expected to occur in any organ with corresponding segmental innervation.

Alexander (1922) described a case with multiple nævi unrelated segmentally to a coexistent vascular spinal cord lesion.

In Rand's case (1927) a pigmented nævus of the skin of the back, at the lower border of the right scapula, led to a pre-operative diagnosis of hæmangioma of the cord, which was confirmed subsequently at operation.

Kaplan (1935) recorded the case of a woman, aged 27, with an extradural hæmangioblastoma, opposite the laminæ of D.5 to D.12, and a nævus just to the left of the 5th to 12th dorsal spines.

Sterling (1936) described a case with a nævus behind the right knee, and a segmentally related tumour of the spinal cord.

Johnston (1938) recorded the case of a boy, aged 5, with a nævus 8 cm. in diameter just below the left scapula over the tenth rib, with symptoms and signs pointing to a spinal cord lesion. A sudden exacerbation of symptoms, due, it was thought, to rupture of one of the thin-walled blood spaces of an epidural hæmangioma, at the level of the 10th dorsal segment, was followed by death. At autopsy an extradural hæmangioma was found, extending from the 9th to 10th dorsal segments of the spinal cord. There was a localized area of atrophy of the cord opposite.

Karshner, Rand, and Reeves (1939) described a case, in a boy aged 14, with scattered pigmented hæmangiomata, especially in the region of the 5th to 9th thoracic dermatomes. A pre-operative diagnosis of vertebral hæmangioma, involving the region of the 7th thoracic vertebra, was made, and on operation a vascular extradural hæmangioma involving the laminæ of the 6th to 8th thoracic vertebræ was exposed. The dura was not opened. Pathological examination of this tumour showed endothelium-lined channels filled with blood-corpuscles, lying in a stroma of connective and fatty tissue. They state that it remains questionable whether vertebral and epidural hæmangiomata represent neoplasms or vascular malformations. These are different from hæmangioblastomata, being relatively benign, slow in growth, and not metastatic, with most of the accumulating evidence suggesting a segmental origin.

Ewing (1940) mentioned a case with an extradural angioma of the spinal cord and angiomata of both breasts, skin, mucous membranes, and lungs. It is not clear whether the skin angiomata were congenital or due to secondary involvement.

Turner and Kernohan (1941) described, among a series of vascular tumours of the spinal cord, an epidural hæmangioma associated with an angioma of the muscles and skin of corresponding metameres. They classify angiomatous growths of the nervous system into true tumours (blastomata) and malformations (hamartomata) though they state that a neoplastic one may have had its origin in the confines of a malformation.

Wyburn-Mason (1943) collected several cases where skin nævi were associated with vascular tumours of the spinal cord. One of these, in a woman aged 22, a case of Jefferson's, had a curious telangiectatic malformation of the subcutaneous tissue, covered in places by a keratinization of the skin (angiokeratoma or angioma verruqueux). The nævi were of two types, one raised above the skin and of an indescribable blue colour, the other flat and more diffuse, forming typical telangiectases. These were confined to the left side of the body and involved three areas—the dermatomes C.8 to D.1, D.7 to D.11, and L.4 to S.3. Autopsy showed telangiectases, mainly confined to the left side of the cord, in neuromeres corresponding to the affected skin dermatomes. He suggests that such metameric nævi may frequently be associated with corresponding lesions of the cord, but are only brought to light when hæmatomyelia occurs, as in this case. He divides lesions into: (*a*) Vascular lesions of the cord, which are either in the nature of telangiectases, cavernomata, or cavernous angiomata, the latter with gliosed tissue between the vessels, or hæmangioblastomata with intravascular endothelial-cell proliferation in the telangiectases (this latter is not usually the type associated with skin nævi); and (*b*) The extradural variety (to which the case described below belongs) —cavernous angiomata, with large vascular sinuses,

Fig. 349.

Fig. 350.

FIGS. 349–352.—Photographs showing extent of the nævus. The operation scar is visible in *Fig.* 352.

Fig. 351.

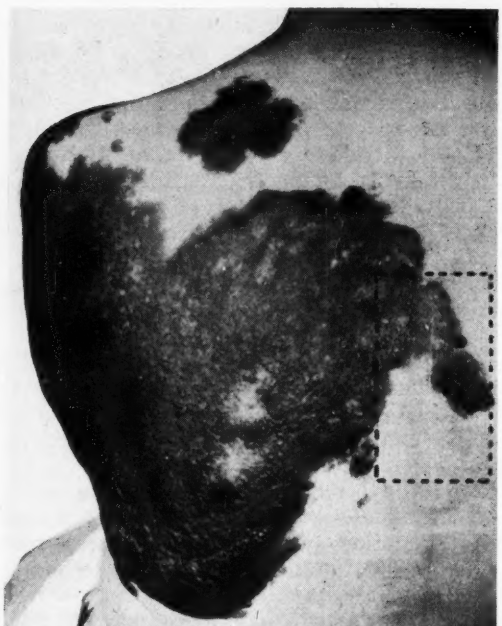

Fig. 352.

the walls being formed by endothelial cells, resting on a very loose connective tissue, which contains variable amounts of fat. His view is that there is no direct connexion between metameric nævi and a spinal vascular abnormality, but that their coexistence is due to a developmental disorder of the whole neuromyodermatome, and that neither of these abnormalities is dependent on the other.

CASE REPORT

HISTORY.—F. G., female, aged 47, developed a gradually increasing paraplegia since December, 1943, both legs being affected equally. She was then resident in London. In August, 1944, in consequence of enemy action, she went to live in the country. Here, whilst using a lavatory, the pan broke, and she sustained an extensive lacerated wound of her left buttock, necessitating the insertion of seven sutures, the wound subsequently becoming septic. Her doctor recommended her to leave the area, and she was evacuated to Birmingham. She did office work right up to the time of the accident. Since, then, however, she has been unable to walk without assistance, or holding on to objects. There was nothing of note in the previous history. Her husband and her two children, aged 24 and 22, are alive

and well, and there is no family history of an extensive nævus or of nervous disorder.

ON EXAMINATION.—

Skin Condition.—There is a congenital purplish discoloration of the skin. This involves the whole of the left arm, forearm and hand, the anterior aspect of the chest, including the breast area, extending from the xiphisternum medially to the 10th rib level in the mid-axillary line below, and to the clavicle above, with, however, a patch of normal skin just below the outer extremity of the clavicle. Posteriorly, it tapers off towards the middle line, where it ends approximately at the level of the 4th to 6th thoracic spines, extending a little over at one place. There is an isolated patch over the spine of the scapula. There are scattered areas of hyperkeratosis, especially in the upper anterior aspect of the chest. (*Figs.* 349–352.) There is some swelling, probably of a cavernous nature, of the fingers, especially the first and terminal phalanges, and of the distal part of the palm (*Fig.* 353).

FIG. 353.—Photograph of hand showing the swelling of first and third phalanges.

Neurological Examination.—Memory for past and recent events was normal, intelligence and speech were normal, and she was of a cheerful, almost euphoric, disposition. Examination of the cranial nerves and nerve-supply to the upper limbs revealed no abnormality. The abdominal reflexes were absent. There was gross loss of power throughout the lower limbs, worse on the left side, with marked increase of tone in both. On testing sensation, there was a poorly defined hypo-algesia, hypo-æsthesia, and hypothermæsthesia up to a vague level about the costal margin posteriorly, and to an even less definite level anteriorly. Proprioceptive sense was absent in the big toes and diminished at ankles. Vibration sense was absent to approximately the level of the 2nd or 3rd lumbar spinous processes. The lower limbs were cold, the feet bluish and œdematous, with a tendency to clubbing. Knee- and ankle-jerks were exaggerated; patellar clonus, exhaustible, was present equally on both sides, as also was ankle clonus, which was of an inexhaustible type. Both plantar reflexes were extensor. Rossolimo's test was negative on both sides. The reflexogenous zone for Babinski's test extended up to the 10th or 11th dorsal segment on both sides. A pin-prick applied to thighs and lower abdomen caused involuntary withdrawal of the corresponding lower limb.

On examination of the spine, there was full mobility and no tenderness.

A presumptive diagnosis of neoplasm of the spinal cord was made.

Cerebrospinal Fluid (Oct. 2) gave the following content:—

Protein	107 mg./100 c.c.
NaCl	734 mg./100 c.c.
Sugar	64 mg./100 c.c.
Globulin (Pandy, Nonne-Apelt)	trace
Lange	1 1 2 2 2 1 1 0 0 0 0

There was an average of 1 cell per c.c. The C.S.F. pressure was 170 mm.; Queckenstedt's test showed a complete block, both 30 mm. and 50 mm. jugular pressure giving no rise in C.S.F. pressure.

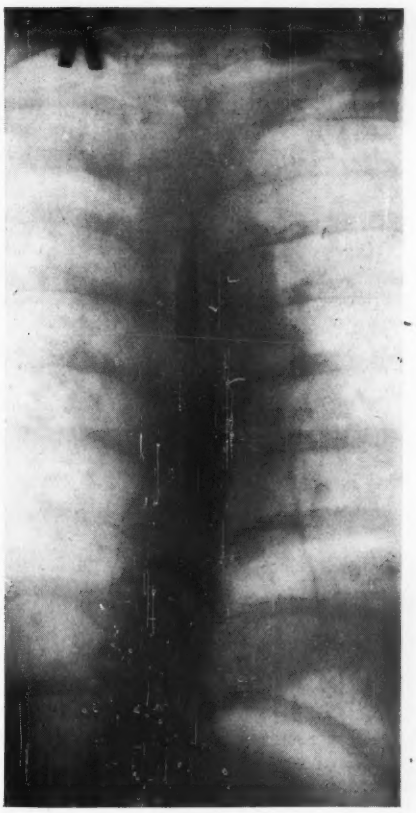

FIG. 354.—Radiograph showing a narrow column of lipiodol in lower and mid-thoracic regions, with widening of column in upper thoracic region. This suggests an extensive malformation of vessels along the thoracic cord, especially on the right side.

A further sample on Nov. 7 contained:—

Protein	165 mg./100 c.c.
Pandy and Nonne-Apelt tests	+ +

Wassermann: Blood and C.S.F. were negative.

Blood-count:

Red blood-cells	5,290,000
Hæmoglobin	108 per cent
Colour index	1·02

Radiological Examination.—Radiographs showed only arthritis of D.10 and D.11 vertebræ, and osteo-arthritis of the sacro-iliac joints.

Lipiodol Injection.—On Nov. 7, 2 c.c. of lipiodol were injected intrathecally into the L.4 to L.5 interspace. No obstruction was detected on screening. On Nov. 12, however, myelography showed a very slow flow of a narrow column in lower and mid-thoracic regions, widening in upper thoracic region, with a suggestion of

an extensive malformation of vessels along the thoracic cord. (*Fig.* 354.)

Course.—On the evening of Nov. 7 patient complained of much pain in the legs and paralysis of legs and toes; there was also dysuria on this and the following day. Muscular power in the legs gradually improved, but she was still unable to walk with assistance, which she was able to do before the lipiodol. A more definite level of sensory loss was now obtained.

On Nov. 22 there was still marked weakness of both legs. She was barely able to do the heel-knee test on each side. There was gross weakness of dorsiflexors in both feet, and of toe movements, left worse than right. There was spasticity of both quadriceps muscles. There was anæsthesia to cotton-wool up to the level of the fifth rib on both sides anteriorly, and up to the eighth thoracic spine posteriorly, with sacral sparing over buttocks only; thermo-anæsthesia and analgesia from L.1 to S.3 on right and L.2–S.3 on left side; hypothermoæsthesia and hypo-algesia from posterior mid-thigh up to and including buttocks on both sides, and anteriorly up to level of fifth rib on each side. (*Fig.* 355.) Rossolimo's test was now positive on both sides. There was normal pin, touch, and thermal sensation over the nævus.

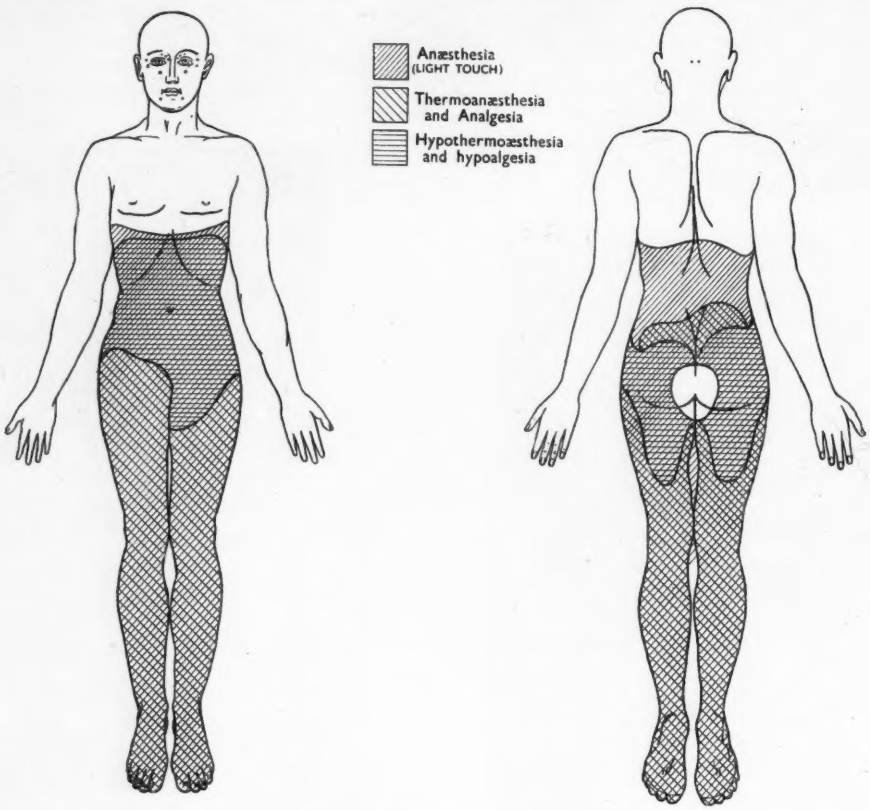

FIG. 355.—Charts showing sensory changes.

AT OPERATION (Nov. 23).—Under intratracheal nitrous oxide, oxygen, and ether anæsthesia, a midline incision was made in the upper thoracic region (*see* scar in photograph, *Fig.* 352) and the laminæ of T.4 to T.7 were removed. This disclosed, immediately beneath, a mass of tangled blood-vessels which bulged into the gap made by removing the bone. From its colour the mass appeared to be exclusively venous and there was no marked pulsation. It extended over three full vertebræ and lay outside the dura, both on the right and left of the middle line. Excision or removal of a portion for biopsy was not attempted owing to the risk of hæmorrhage. It was thought that the vascular malformation was in a type of vessel which might well respond to X-ray treatment. Hæmostasis was therefore secured, and the wound sutured with silk, in layers, two for the muscle and one each for the deep fascia, superficial fascia, and skin, without drainage, sulphanilamide powder being incorporated in the layers of closure. Patient was shocked and rather cold after the operation, but soon picked up under the radiant-heat cradle. Healing of the wound was uneventful.

A course of deep X-ray therapy to the mid-dorsal region followed.

A radiograph taken some time after the operation, with the patient tilted, showed that although there were still droplets of lipiodol present, none was present above the 9th dorsal vertebra (*Fig.* 356). No abnormal calcifications were present. A radiograph of the chest showed no definite lesion or abnormal vascular markings.

She had œdema of ankles and lower half of legs with trophic changes in both feet for a time, but these gradually subsided and sensation began to return, until, on May 2, 1945, sensation to pin-prick, cotton-wool, thermal, and vibration sense was apparently normal. There was still, however, some disturbance of joint sense. She could stand without assistance, which she was not able to do previously, but was still unable to walk without assistance or holding on to objects. In a few small scattered areas, the skin of the forearm became less deeply pigmented, in contrast to the remainder of the skin in this area.

DISCUSSION AND SUMMARY

A case is described, in a woman aged 47, of a vascular extradural spinal cord tumour, at the level of the 6th to 10th thoracic segments, whose presence was confirmed at operation, with also a very extensive

congenital nævus, involving the 5th cervical to 10th thoracic dermatomes on the left side.

Symptoms commenced eleven months before operation, with acute exacerbations eight months later, and a fortnight before operation, the latter

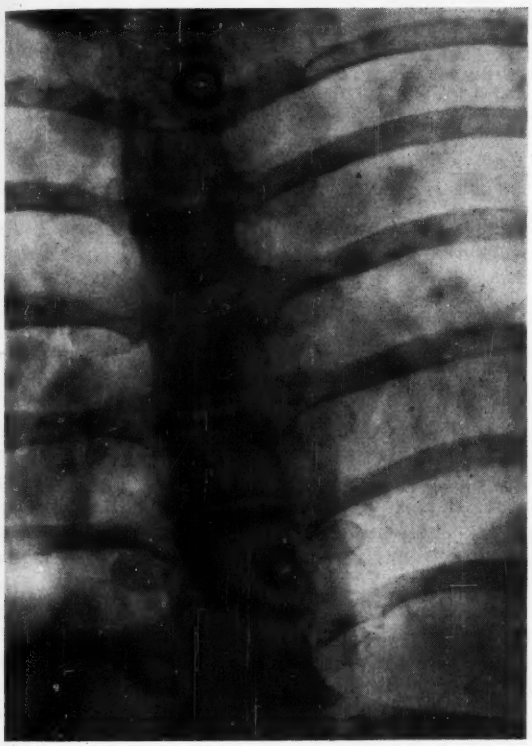

FIG. 356.—Anteroposterior radiograph of the thoracic and upper lumbar spine, showing absence of laminæ of T.4–7 and lipiodol up to T.9 only.

following lipiodol injection. Discontinuity and progression by a series of apoplectiform stages or 'episodes' is characteristic of vascular tumours of the spinal cord.

The possibility of the presence of a vascular abnormality in the corresponding segment of the spinal cord should be kept in mind in patients presenting themselves with signs and symptoms of a spinal cord lesion when there is a nævus of the skin. Though malformations of the spinal cord, due to a disturbance of mesenchymal development, similar to and usually in the same segment as that of the skin condition may be present, their presence may not be suspected until either hæmorrhage occurs or they increase in size, at any time, as in the case described.

The cases recorded in the literature are reviewed; most of them are of a simple hæmangiomatous nature, corresponding to the skin condition, rather than hæmoblastomatous.

My thanks are due to Dr. Williams-Walker, under whose care the patient was admitted to the Midland Nerve Hospital, for his kind assistance in the preparation of this paper; to Mr. W. H. Sweet, Neurological Surgeon to the Hospital, who performed the operation, and who kindly allowed me to use his examination and operation notes; and to Dr. J. F. Brailsford for the X-ray examinations.

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THE MANAGEMENT AND SURGICAL RESURFACING OF SERIOUS BURNS

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THIS paper is based on the management of a series of consecutive serious burns and the resurfacing problems presented by 192 of them.

The immense number of burns in this war has been chiefly due to petroleum products, phosphorus, and cordite, which in that order are the commonest causes of military burns. The problem which a burns casualty presents to the surgeon is a different one in each of the three phases, early, intermediate, and late, through which all serious burns pass. In each of the three Services, too, the burns problem has its own emphasis.

The Navy is faced often with the task of providing primary treatment for a large number of serious burns at the same time, and often under the most difficult circumstances. The R.A.F. have from the first had a special interest in the burns of exposed parts of functional importance—the hands and face. No special type of burn is peculiar to the Army, but Army hospitals, especially those in the Mediterranean theatre, probably see more of the extensive petrol burn with very large areas of skin destruction than are seen elsewhere; the majority of these are the result of accidents.

Treatment in the primary phase of a burn is concerned chiefly with the preservation of life and prophylaxis of infection. The majority of military burns have their primary treatment at Casualty Clearing Stations in quiet periods, or at General Hospitals. The theatre treatment of the battle casualty burn must be simple. It consists usually of "toilet, S.V.G.". This is effective for most superficial burns, and little later treatment is needed. When the full-thickness of the skin has been destroyed, the primary phase is followed by the intermediate and late phases of burns treatment with which this paper is concerned.

The intermediate starts in the second week. It is the period of healing of the burn, and comprises the preparation of the patient and of the burnt area for grafting, and the application of grafts and their care until healing is complete. Surgery in the late phase, that of rehabilitation, is necessary only for that minority of burns which primary cover does not give the best functional result. The ideal treatment is to cover the raw area as early as possible with new skin which will meet the full functional and cosmetic need of the part; but a granulating surface will not always accept a thick free graft, and a thin graft always contracts later. Because of this, some areas such as hands and face must be treated in two stages; a primary cover is provided by thin free grafts which are later excised and replaced by thicker grafts or flaps to relieve the disability of the thin tight primary cover.

Material on which this Paper is Based.—For two years all serious burns reaching Main Base (first in North Africa and later in Italy) were referred to this unit; 800 cases were treated. Of these, nearly 25 per cent involved the full thickness of the skin and were grafted. This paper describes the management of these 192 cases and the resurfacing problems of their intermediate (or healing) and late (or rehabilitation) phases.

The policy of the unit is to retain all cases until healed; each case is then assessed with reference to his capacity for duty in the Mediterranean theatre. Sun sensitivity of recently healed burnt skin is common in the Mediterranean and makes many cases unfit for further service there for at least 3–6 months. Thus only 16 per cent of the 192 cases with full-thickness skin destruction returned direct to duty in this theatre.

The burns came from all three Services, but Army personnel accounted for 80 per cent. Over 80 per cent of all cases had their primary treatment elsewhere. In North Africa the time of arrival of cases varied between 1 week and 6 months; in Italy the interval has been more uniformly 1–3 weeks, but in a number of cases was 8 weeks or more.

Of the 150 cases admitted to the Unit within 24 hours of being burnt, nearly 100 came from naval incidents caused respectively by phosphorus, cordite, and superheated steam.

Table I shows the incidence of causes of burns with full-thickness loss. The battle casualties are higher in this group (40 per cent) than in the total burns, of which they constituted only 30 per cent. The reason for this is that phosphorus, shell, and grenade burns almost always destroy the full thickness of the skin, and in tank burns there are often associated wounds and delays in treatment which predispose to secondary infective loss.

Table I.—Incidence of Causes of Burns with Full-thickness Loss

Accidents:		
Petrol	45 per cent	59 per cent
Other	12 „ „	
Electric	2 „ „	
Battle Casualties:		
Phosphorus	16 per cent	41 per cent
Tank	11 „ „	
Cordite	6 „ „	
Petrol (battle)	6 „ „	
Chemical	2 „ „	

Table II.—Extent of 192 Burns with Full-thickness Skin Loss

Per Cent Body Area	*Per Cent Incidence*
0–10	28
10–20	32
20–30	18
30–40	10
40–50	5
50–60	5
60–70	1
70–80	1

The extensive nature of most of the cases in this series is shown in *Table II*. Seventy-two per cent involved over 10 per cent of the body surface (that is, more than half the whole lower limb; or both hands and head and neck). In 20 per cent of the cases one-third or more of the body surface was burnt. All these were Army personnel and almost all of them were treated in Italy. In North Africa very few burns of over 30 per cent body surface reached Algiers from Tunisia. Everyone of the 13 cases which involved more than 50 per cent of the body surface was caused by petrol.

Table III.—Analysing 192 Cases according to extent of Full-thickness Skin Loss

Area of Full-thickness Skin Loss	*Percentage Incidence*
sq. cm.	
0–100	16
100–300	34
300–500	15
500–1000	12
1000–2000	17
2000–5000	6

Table III classifies the cases in this paper according to the extent of the full-thickness skin loss. This classification has been made without reference to the site, as the problems of resurfacing special areas are considered separately. A primary concern of this paper is to show that the size of the granulating area is a major factor in the programme of treatment, but not in the time taken to heal it. The cases described here are predominantly extensive areas of skin loss: 68 showed over 500 sq. cm. loss. In 46 the loss of skin was over 1000 sq. cm.; that means, for example, almost total loss of the whole skin on a normal-sized thigh with the exception of a strip about 10 cm. wide along the outer side, where the skin is thickest; or the whole of the back and parts of the sides of a thigh, popliteal fossa, and calf; or most of the back from the base of the neck to the lumbar region.

The clinical problem in these extensive cases is far wider than that of grafting alone. Almost every patient with 1000 sq. cm. or more of raw granulating

surface is constitutionally ill until his raw area is substantially reduced below this figure. The upper limit of granulating area compatible with life is not known, but in this series there were 4 cases with 3000–5000 sq. cm. granulating, all of which survived.

The incidence of severe complicating conditions in these cases was high. The 46 cases with over 1000 sq. cm. of full-thickness skin loss included 1 with severe typhoid, 4 with infective hepatitis, 6 with severe malaria, 1 with acute glomerulo-nephritis, 1 with glandular fever, 5 with blast lung, and 3 with fractured femur.

PRINCIPLES OF TREATMENT

General Treatment.—The main purpose of early treatment is to restore circulation. This is done chiefly by liberal administration of fluids. Progress is controlled by 3-hourly hæmoglobin, blood-pressure, and pulse records for the first 24–72 hours, and by fluid balance sheets.

Hb of 140–150 per cent is not uncommon a few hours after severe burns and the systolic pressure may be 40 mm. of mercury or below. There is evidence, which our material confirms, that this combination can cause renal anuria which never recovers, though the B.P. and Hb are returned to normal within a few hours, by prompt energetic treatment. This consists of fast plasma transfusions which are slowed down when the Hb returns to 100 per cent. The patient is usually thirsty and should be given plenty to drink.

Within a week profound anæmia commences and is treated by repeated blood transfusions of 1–2 pints several times a week, aiming at an average Hb of 80–90 per cent. In cases of estensive skin loss it may be impossible to reach this Hb per cent, however intense the transfusion programme, and improvements following transfusion are likely to be temporary. With healing of the granulating surfaces by grafting the patient becomes independent of transfusion.

As soon as they can take it, the patients are put on a special high-protein, high-sulphur, high-calorie diet, consisting largely of steak and eggs, with champagne, brandy, ice cream, and iced fruit juices. Additional iron and vitamin C are given as medicines. Neglect and ignorance are the only causes of gross emaciation in burnt patients. They are often helpless and incapable of feeding themselves and must be as patiently and attentively fed as they are nursed. Given a full, palatable, and balanced diet with a rapid resurfacing programme emaciation does not occur. Once they are healed the protein and nitrogen loss ceases and a good appetite restores the metabolic balance.

It has been our practice to use penicillin only for a definite purpose, such as the preparation of the surface for grafting or the treatment of some susceptible complication.

In summer flies and heat are a problem. Apart from the discomfort they cause, they both threaten life and are controlled only by air-conditioned, fly-proof wards. It is also important not to overheat the patient by covering him with cotton-wool dressings from head to foot. As soon as the patient is fit enough, a physiotherapist starts work to encourage recovery of function and prevent contractions. Hands, neck, and eyelids are the most important objectives at first; and later large joints and re-education in walking. The saline bath is very valuable in re-establishing limb movements in extensive burns.

Evacuation and Travel of Burns.—Burns travel well in the first few hours and all considerations should be subordinated to getting them within 6–12 hours to a General Hospital which can hold them, and give them full services.

If early evacuation is not possible the important thing to realize is that, after their first theatre treatment and anæsthetic, severe burns travel very badly and must be held for several days. Most severe cases are quite unfit for any journey by air or ambulance for a week after operation, and continue to be so, long after they are in need of the full facilities of a General Hospital. The choice of time of evacuation to a General Hospital can be one of great difficulty and ultimately depends on the response of the individual burnt; but any serious case and all affecting 30 per cent area of the body should be held forward for 7–10 days after primary theatre treatment. Circulatory collapse during transit is common in the ill cases and, if they have to be evacuated, they should have a plasma drip running under supervision.

Local Treatment.—The standard local treatment of all cases in Italy and most of those in North Africa, has been a dressing of S.V.G. after an initial gentle toilet with removal of dead skin, generally after pentothal anæsthesia. The method cannot be called ideal; there are serious objections to swathing a patient in vaseline and wool, particularly in hot climates; fluid loss from the burnt surface is not controlled and bacteriostasis is incomplete and not persistent. In phosphorus burns there has been evidence that early removal of the particles and neutralization has averted full-thickness skin loss in some cases. We have not used the method of cotton-waste compression.

SIGNIFICANCE OF DEPTH

Much of the confusion of thought that envelops the subject of healing of burns arises from failure to make the critical diagnosis between burns involving partial skin thickness and those with destruction of the whole thickness of the skin. The former may be expected to heal naturally in 1–5 weeks, the time depending mainly on the depth to which the skin is burnt. The latter, however small, are almost invariably best treated by a skin-graft, and it must be clearly realized that an indecisive expectant policy in these cases delays healing inexcusably and often causes permanent crippling or death to the patient.

The management of a burnt patient has thus two main aspects: first, the metabolic problem; and, secondly, the diagnosis of full-thickness skin loss. Early diagnosis of full-thickness skin loss is imperative because once it is made, all attention must be focused on preparing the patient and the surface for skin-grafting as early as possible. It is well here to state the the time-table to which local and metabolic treatment must be geared, so as to shorten the painful and dangerous stages to the minimum, by achieving rapid surgical resurfacing; twelve to eighteen days after burning the areas of full-thickness

skin destruction can be accurately assessed and the sloughs excised under anæsthesia. Two days later, close-packed, thin patch grafts are applied and one or two further applications of patch grafts may be needed for extensive burns at two- to three-day intervals; three to seven days after the last grafting operation the patient is healed—that is, on the twentieth to thirty-fifth day after injury. This rapid time-table can be followed in the majority of cases, including many very extensive burns, and is the standard to be aimed at always. As explained below, however, it requires close team-work and supervision, and in a proportion, particularly of complicated cases and those seen later, primary resurfacing is not complete until forty to sixty days.

CLINICAL DIAGNOSIS OF DEPTH

In taking the history it has been our routine to make notes of the agent and circumstances causing the burn, to record what the patient was wearing, and if his clothes caught fire. It is not surprising that patients' estimates of the length of time that they were being burnt are erratic, but it is generally true to say that a petrol-vapour explosion lasts only a second or so and causes only epidermal burns.

The flash of burning cordite may last up to seven seconds and usually causes dermal burns with patchy full-thickness loss. Burns with large areas of full-thickness loss generally give a history of prolonged exposure to the flames—roughly thirty to three hundred seconds—due to being splashed with flaming petrol, trapped in a blazing tank, wearing petrol-soaked clothes, or being unconscious or helpless at the time of the burn. These are cases of whole-thickness skin destruction caused by the initial severity of the burn.

Phosphorus particles from incendiary weapons typically cause sharply demarcated scattered areas of full-thickness loss, or sometimes larger areas if the clothes catch fire.

Even a single layer of clothing gives good protection from burning, but if the clothes catch fire, full-thickness burns generally occur, unless they are taken off immediately. In contra-distinction to pilots, tank crews wear little protection from flames. For this reason unnecessarily severe burns of hands and face are common amongst them.

In the Mediterranean theatre the difference between summer and winter clothing produces an interesting seasonal variation in the incidence and distribution of the burnt areas. In summer it is common to see a burn confined by a sharp boundary to the parts left uncovered by shirt, shorts, and hose tops. The clothes did not catch fire and there is no doubt that gloves and battle-dress would have completely protected all except the face; but when soaked with petrol, a battle-dress burns fiercely, it is difficult to take off in a hurry, and typically causes very deep burns of the entire trouser area.

Electric and hot-water-bottle burns have always involved full-thickness skin destruction.

Some idea of the depth of a burn may thus be gained from the history, but accurate assessment always depends on recognizing the appearance of burns of different depths. For simplicity we recognize three chief depths of burn, each of which is distinct in prognosis and treatment.

Epidermal (E.B.)—This is the burn caused by minor scalds and momentary explosions; a stage of erythema is generally followed in a few hours by the appearance of lax superficial vesicles or bullæ of serum. Under these is the familiar red, exudative, painful skin. Treatment is aimed at comfort and the avoidance of bacterial infection; the burn is dry within a week and leaves no permanent mark on the skin.

Full-thickness Loss (F.T.L.).—This term is self-explanatory. In the grossest examples the skin may be initially charred, and in these cases there is invariably heat coagulation of deeper structures; but usually the heat has been sufficient to produce coagulation necrosis of the whole thickness of the skin, and not intense enough to dry it up. Commonly the dry epidermis may be wiped off exposing the underlying parboiled skin. It is most important to realize that it is not possible to diagnose the exact area of full-thickness destruction from this appearance. The parboiled skin is at first a cyanotic red colour or dead white and insensitive to touch, in striking contrast to the raw area of E.B. Within a few hours it is surrounded by gross inflammatory subcutaneous œdema, and in a few days it becomes yellowish and the central parts tend to dry up to form a dark-brown leathery slough. Deep to this and at the margins, a plane of separation appears by about the twelfth to eighteenth day and the slough loosens by phagocytic digestion, which causes the accumulation of a lake of pus beneath the slough. The retention of this pus in contact with a large raw area is responsible for the toxæmia and fever which occur at this stage, and it is obviously beneficial to hasten removal of the sloughs surgically.

Within two days of early excision of the sloughs the surface of the subcutaneous fat becomes firm, red, and finely granular, and is ideal for grafting. If the sloughs are allowed to separate naturally they may take twenty-five to forty days and then a smooth glistening gelatinous resurface, with a fibrous reticulum is left. This is best prepared for grafting by daily hypertonic saline dressings, but if treated by infrequent dressings or encased in plaster, the granulations hypertrophy and project above the surface of the skin as a gelatinous friable sheet which is not a sound bed on which to apply free skin, but is usually best removed by abrasion under anæsthesia before grafting.

Dermal (D.B.).—This group is intermediate between the two former and includes all burns in which skin destruction extends through the malphighian layer into the dermis, but not deep enough to cause total epithelial destruction. It is a clinical group of great importance. At first, the appearance of the cyanotic or white layer of shiny moist skin beneath the epidermis may be identical with that of F.T.L., and though patches of F.T.L. may be rightly suspected, they cannot be accurately mapped out until the separation of a thin and sometimes cheesy slough commences at about five to ten days. The surviving gland and hair follicles then give the surface a stippled appearance which is characteristic and the skin may be expected to heal naturally three to five weeks from the date of burning. In the deepest burns of this group, survival of the epithelial foci is precarious; infection of the area may convert

it into one of full-thickness loss and must be avoided by frequent dressings with scrupulous care. It is our impression that secondary infective full-thickness skin loss is common, but that it usually occurs in cases where skin loss requiring a graft is already present and in not more than 1–2 per cent of burns that are initially part thickness in depth.

The healed skin of dermal burns is thin and delicate, often has an irregular surface, readily vesiculates and becomes abraded or cracked. It is itchy and very sensitive to sunshine and has a sluggish circulation, especially in dependent extremities in cold weather. It is at first very vascular but later pales.

During the first few months after healing, the skin becomes thicker, chiefly by the deposition of a dense dermal fibrous layer. This process is accompanied by a strong tendency to contraction of the scar, which may cause serious deformities and limitation of joint movements, or may convert the affected part into a tight stiff plaque.

In the course of time the layer softens, but the skin never returns to its normal thickness and elasticity. Pigmentary changes and sensitiveness to sunlight may remain for life.

For these reasons dermal scars in some sites may require late plastic repair.

PREPARATION OF THE SURFACE

The object is to make all areas of full-thickness destruction ideal for grafting as soon as possible.

As soon as the area of F.T.L. can be accurately assessed—generally early in the third week—it is to the patient's advantage to have all sloughs removed from the area by scissors and knife. Hæmostasis is then obtained by hot flavine-gauze pressure dressings, and on their removal under a further anæsthetic two to four days later, the surface is ideal for grafting. It is a firm, flat, smooth, red bed which bleeds slightly on removal of the dressings. If light insufflation with penicillin powder or intramuscular penicillin is used, the surface is even more receptive and the take of the graft is almost invariably 95 per cent or over.

Operative removal of the sloughs is not often practicable before 12–18 days, and the earlier it is done, the more hæmorrhage and trauma it causes. Excision of sloughs from very large areas (2000 sq. cm. and over) may cause serious circulatory collapse and should only be done in these cases by an operating team and anæsthetist with experience in assessing their fitness, and with a good blood service. Where it can be applied on sound skin a tourniquet will reduce the amount of blood lost during the operation. Closed-circuit cyclopropane anæsthesia is very suitable and safe. The operative method requires a minimum of ward work; the preparation of the surface for grafting is done in the theatre 2–3 weeks after the date of burning, daily ward dressings are needed for about a week after the final graft, and the patient is then healed and ready for evacuation, or whatever his correct disposal may be. This time-table heals a burn with maximum certainty in a minimum time—about twenty to thirty days from injury; but some of the most severe and complicated burns are too ill to stand it and must be handled carefully at a slower pace. The method is economical of hospital beds and skilled ward care, but it demands close supervision by the surgeon and the additional burden on the theatre may make it impracticable. For example, at one period of peak pressure of maxillo-facial work, there were also 90 serious burns in the wards.

The second principal method of preparing the surface is the non-operative one and consists in assisting the natural separation of the sloughs. Its particular value is to relieve the theatre at exceptionally busy periods, and in these circumstances it has largely been entrusted to the ward staffs. The result of this method is undoubtedly that grafting and healing are delayed and the skin is less stable, and each case in the end takes twice as much work to heal it. The method demands great patience and skill and needs a large ward staff. The burn is dressed daily or more often with meticulous technique and at every dressing all marginal crusts, discharges, dead matter, and loosening sloughs are carefully removed. The surface is wiped dry with gauze pledgets, bathed with flavine solution, and covered with a single layer of vaseline-gauze (to prevent pain in removing the dressing). Outside this is a thick layer of gauze soaked in sod. sulph. 12 per cent in flavine 1–1000, and pressure applied with a crêpe bandage over wool. This method produces a firm layer of flat granulations, with little discharge and suitable for grafting, in two to five days or as soon as the sloughs are off—twenty-one to thirty-five days from the burn. A daily warm normal saline bath to soften the sloughs and assist the separation is of great value. Its proper management is a highly skilled technique and to some extent a physical ordeal, for in badly burnt patients it occasionally causes collapse. Daily light insufflation of the surface with penicillin powder shortens the time required to prepare the surface for grafting and increases the percentage take by reducing the discharge and removing the hæmolytic streptococcus.

When cases of very extensive burns are first referred for grafting long after the separation of the sloughs, and the granulations are hypertrophic and œdematous, the same method applies; but as they have generally also had a long period of malnutrition and toxæmia, it is often wiser to accept the disadvantages of preparing the granulations by hypertonic dressings, than to endanger life by excising them. In many smaller areas, however, the treatment by radical excision or abrasion is better and without risk.

The operative and non-operative methods have both been used in the series analysed here, and several times the two methods have been used in different parts of the same raw area, so that exact comparisons have been possible. The over-riding reason for preferring the radical theatre preparation is that it is short and painless.

CHOICE OF METHODS OF PRIMARY GRAFTING

It is widely accepted that in grafting granulating areas there is a risk of failure. This can be reduced by correct preparation of the surface, so as to reduce discharge and infection; it is equally important to determine the best method of skin-grafting. The

types of graft under consideration are patch, sheet, and pinch grafts.

It is found in practice that the thinner a split skin-graft is, the more likely it is to take on an unfavourable surface. At first a free graft is nourished by direct diffusion from below; in some cases this may be enough for the whole thickness of a thin graft to live, yet if the graft were thicker, only the deeper layers would survive. This conception explains the surface necrosis and desquamation sometimes seen in thick free grafts, and the reliable take of thin grafts.

Patch Grafts.—For very large areas that must be covered as quickly as possible by a simple safe operation, multiple thin patch grafts are at present the best method. A certain take of 90–100 per cent can be obtained even on an indifferent surface.

The success of patches makes it reasonable to expect that a primary cover by suspension of minced skin may become practicable.

Sheet Grafts.—For limited areas requiring strong supple skin a 'primary-final' repair may be obtained by a successful sheet graft; but it must be emphasized that there is a tendency for late contracture in all grafts placed on granulating surfaces, that the method calls for a higher standard of preparation and technique than when patches are used, and that it is impracticable to cover some of the largest burns by sheets because of the amount of donor skin required. A 50 per cent gum acacia glue applied to the raw surface of the graft in a thin film is used to secure it and promote a good take. It is as effective as Sano extracts and less trouble to prepare.

Pinch Grafts.—Pinch grafts have no place in this scheme. They can undoubtedly take readily and need no skill in application, but in every instance patch grafts are better. Patches produce quicker and more certain healing of better quality and appearance, and the donor areas are practically without scar and can be used again and again.

OPERATIVE NOTES

Cutting and Application of Patches.—The two chief graft-cutting instruments are the Padgett dermatome and the Blair knife. The dermatome is almost indispensable when the usual donor sites are not available. It is also very suitable for cutting large thick sheet grafts of uniform depth in late repairs; but for patch-graft cover of granulating burns, hand-cut skin is preferable as the dermatome cannot cut the skin as thin as is desirable, leaves scars which take longer to heal because they are deeper, and the dermatome cannot use a donor-site as completely at one session as can be done by hand. For example, we cannot cut skin thinner than $\frac{5}{1000}$ in. satisfactorily with a dermatome, and at that thinness the graft is difficult to handle and generally tears during removal from the drum. With a Blair knife, it is easy to cut 1000 sq. cm. of skin from one thigh at one operation; but it would be very difficult to cut the same amount from one thigh—the equivalent of five full drums—with a dermatome. We believe that the best skin for patch-grafting granulating areas is the thinnest that can be cut. Very thin patches coalesce more rapidly and the scar is more uniform than when thick ones are used. The durability of the graft depends less on the thickness of the patch than on the intervening areas. That is why it is desirable to graft on a firm flat bed and to place the patches so close that they coalesce within three to seven days.

Sheets of split skin are spread on vaseline paper, cut in strips and then into patches of 1 sq. cm. or less. They are best placed so close together that they are touching; when there is not enough skin for that, they should be cut very small and spread uniformly over the area.

When a dermatome is used as patches on greased paper, it is unnecessary to remove the cement from the sheets as the paper can be removed easily on the fourth day without tearing the grafts. In extensive areas 3–700 patches may be cut and applied at one session. The time taken to apply such a large number of patches can be considerably shortened if a third person is washed-up, and this extra help should be made available for ill cases. The operation then takes about 100 minutes for some 400 patches.

It is a wise plan to cover approximately 1000 sq. cm. at each operation by closely-packed patches. For areas larger than this the operations are staged at 3- to 7-day intervals and are repeated until it is certain that healing will be complete in 10 days.

On the limbs and on most parts of the trunk, these patches are fixed only by a vaseline gauze layer over which flavine-gauze, wool, and crêpe are applied in that order. Fixation of the axilla is a special problem. Here some degree of graft loss due to slipping of the patches has often been seen. Tulle gras fixed to the skin by stay sutures and compression over flavine wool has been found the most satisfactory solution.

Anæsthesia.—Cyclopropane has been used for all the ill cases since it became available. It has been very nearly ideal with seriously ill patients and they recover consciousness completely two minutes after a two-hour operation.

Continuous pentothal is contra-indicated for all cases of severe burns, most of whom have some degree of liver damage. When this method was used it caused profound post-operative coma and hyperthermia in several cases. One such case died. No harm has ever followed induction with 0·5 g. of pentothal or less.

PRIMARY COVER OF SPECIAL AREAS

There are certain sites where patch grafting is inapplicable as a primary cover and others where it is not the best method. In sites such as eyelids, the hands, and the flexor creases, sheets have been used. They are cut thin and well incised. Incisions are made by uniform stabs with No. 11 or No. 15 B.P. blade when the graft is spread on the board.

Eyelids.—In burns with full thickness of the skin of the lids with dry corneæ the need for tarsorrhaphy to prevent corneal ulceration is urgent. In this series 8 primary repairs of lids for such cases were done after the fourth week, and while the lids were still granulating. The lids were mobilized, tarsal borders brought into apposition, lateral and medial tarsorrhaphies performed and the raw area covered by a thin split-skin graft. In every case the tarsorrhaphy held and the grafts were 90–100

per cent take. Although this operation keeps the cornea moist and preserves vision, the cosmetic appearance of thin split-skin grafts is inferior to that which can be obtained later by full-thickness post-auricular grafts.

Hands.—The skin loss in burnt hands demands replacement within 3–4 weeks of burning if contractures and fibrous and secondary necrosis are to be prevented, and if healing is not to be indefinitely delayed. No attempt has been made at primary excision of destroyed skin because of the difficulty at this stage in ascertaining early what is dead, especially in burns affecting tendons and joint capsules. With efficient chemotherapy delay of excision of sloughs for a fortnight carries no risk of secondary infective loss. Between the second and third week a thorough excision of all dead tissue is done under a pneumatic tourniquet. This is followed by 3–4 days' light compression and elevation of the limb, with a full course of parenteral penicillin. The raw area is then covered with well-incised dermatome sheets cut at $\frac{8}{1000}$–$\frac{12}{1000}$ in. and fixed with gum acacia. Active movements are started a week after grafting.

There is no doubt that sheets are the best primary cover for hands, but for certainty of take in primary operations they must be cut thin. These thin sheets undergo considerable contraction which may necessitate later replacements by thick ($\frac{30}{1000}$ in.) dermatome sheets which contract less.

The primary repair of a burnt hand by a sheet is a specialized job, and considerable experience in cutting and fixing of grafts is necessary for a consistent standard of success. The implication of this is that an alternative simpler method can be accepted under certain circumstances because of the absolutely necessary need of providing a skin cover within a month in extensive deep burns of the hands. For this reason there is a role for patches in the primary repair of hands. They are an almost certain method of providing a cover within 7–10 days of the graft. Like thin primary sheets they have later to be excised and replaced by thick skin before full function can be regained; but once a hand has been given a substantial skin cover the prevention of deformity by using a properly constructed splint at night is a simple matter. Supervised active movements during the day can be started within a week of grafting.

Flexor Creases (Popliteal and Cubital Fossæ).—The healing of patches across the flexor creases may be unstable. For this reason sheets of $\frac{10}{1000}$–$\frac{30}{1000}$ in. have been used to cover popliteal and cubital fossæ even when contiguous raw areas were covered by patches.

LATE DEFINITIVE REPAIRS

Late repairs formed only 10 per cent of the operations in this series. They were short-term operations for cases likely to return to duty in the Mediterranean theatre within three months of primary healing. These operations were done for the repair of hands, eyelids, and noses, and consisted of: thick dermatome sheets ($\frac{24}{1000}$ in. and thicker) for faces, hands, and popliteal fossæ; post-auricular Wolfe grafts for eyelids and eyebrows; rotation flaps for axillary and neck scars; and forehead flaps for noses.

LOCAL COMPLICATIONS

The complications to be considered are infective, cicatricial, and dermatological.

Infective Complications.—

Early Infective Graft Loss.—In North Africa the hæmolytic streptococcus was by far the commonest cause; it accounted for partial and occasionally complete graft loss in 15 out of 119 operations (12 per cent). Shortly after the Tunisian battle, there were 10 cases in our burns ward which were heavily infected with sulphonamide-resistant hæmolytic streptococci and had been unreceptive of free grafts despite repeated radical surgical attack. All showed a very poor marginal epithelial ingrowth, which is a most important factor in assessing whether free grafts are likely to take well. When a brisk epithelial response is present at the margin of a burn, grafts may be expected to take, even though cultures persistently show hæmolytic streptococci. Most of these burns were cleared of streptococci by local penicillin when supplies were available, but 5 were evacuated to U.K. still incompletely healed. In Italy, since the routine use of local penicillin, streptococcal infection has ceased to be a major concern. All cases admitted with streptococcal infection present have been cleared of it after two or three daily applications of penicillin, and the burns ward has been completely free from streptococcal infection for weeks at a time.

Use of penicillin has raised the expectation of over 80 per cent take of grafts from 84 per cent (119 cases) to 96 per cent (176 cases), a figure which is capable of further improvement by still greater pre-operative care.

About 10 per cent of the North African burns in this series were infected with morphological K.L.B. None showed systemic toxicity, but two shell wounds infected with morphological K.L.B. which were under our care had peripheral neuritis; pathogenicity tests were not available. At one time four K.L.B.-infected burns showed a late progressive rot of grafts which had initially taken well. All were given 40,000 units of A.D.S.; after this, fresh skin-grafts were applied, took well, and remained stable.

The other organisms commonly found in burns —*Ps. pyocyanea. B. proteus*, staphylococci, and diphtheroids—are all controlled by the régime of frequent ward and theatre toilets and hypertonic pressure dressings, and have not appreciably affected the take of our grafts.

Late Graft Loss—A major unsolved problem in the treatment of these extensive full-thickness burns is a tendency for small areas to break down late and remain as chronic slow-growing superficial ulcers. They often start from abraded blisters and small raw areas and occur most commonly on the leg when early walking has been allowed without support to the grafted areas; they are not particularly common in flexures. No specific organism can be considered responsible, nor has mycotic infection been demonstrated.

The cases that are most subject to them and which are most resistant to treatment are those

which have not been healed promptly by early skin grafts, or where grafts have been applied to a surface of hypertrophic granulations or have been placed too far apart.

This is the main reason for early and intensive grafting of all raw areas and regrafting of any residual area that is not certain to be healed within 10 days.

Patch grafts will sometimes take on these areas after a period of intensive ward and theatre toilets, but if they fail, the raw area and the thick underlying fibrous layer must be totally excised and the fresh bed so prepared can be successfully grafted.

Perichondritis.—This important late complication occurred in 5 per cent of the burns with full-thickness skin loss in this series. It is exquisitely painful and tends to persist until most of the auricular cartilage is destroyed. It is treated by drainage of pus and removal of infected cartilage by curette. Even in cases which respond to one operation there is considerable deformity of the ear on healing, and usually all that is left is a bulbous stump of an ear totally lacking the auricular cartilage.

Cicatricial Complications.—This note concerns the contractures and hypertrophic scars which are seen in the deeper partial-thickness burns, with destruction of the epidermis and superficial parts of the dermis, after healing has occurred from surviving dermal elements within the area of the burn. Such burns take 3–6 weeks to heal. The scars are then red and hypertrophic with considerable underlying dermal fibrosis and the end-result can be as crippling as the scar of a burn with F.T.L. which has healed by marginal ingrowth and contractions.

In sites such as the neck, axilla, and popliteal fossa the scars of dermal burns often cause major persistent disability and are tender, irritable, and tend to crack and ulcerate with minor trauma. This condition has been treated by excision and free sheet grafts. Post-operative X-ray therapy would have been valuable in limiting contraction of the scars. There is probably a role for primary excision of localized dermal burns in these sites with immediate application of free grafts.

Dermatological Complications.—

Pyoderma.—It is quite common for a patient with healed burns to develop a localized or generalized pyoderma, a complication which is resistant to treatment and entails prolonged hospitalization.

Sun-sensitivity of healed burnt skin is common in sunny climates and consists in most cases of mild vesiculation. Free grafts of normal unburnt skin are not subject to it. Acute sulphonamide photosensitivity is a distinct condition and has been seen three times in burns cases. Both these sensitizations are usually temporary.

This group of complications emphasizes the need for treating burns in close collaboration with a dermatologist.

RESULTS

Healing times of the 192 cases grafted are shown in *Table IV*. These times are given from the day of grafting rather than the day of burning because of the very variable times at which cases were admitted. The cases are divided into five groups according to size of area grafted, from less than 100 sq. cm. to over 1000 sq. cm.

A comparison of the overall average healing times of the different groups shows a striking fact. It is that size of granulating area has little relation to time of healing. Thus the average healing time of all areas under 300 sq. cm. was 17 days and that of all the areas between 500 and 1000 sq. cm. was 22 days.

Table IV.—Analysis of Healing Times in 192 Cases Treated by Skin-grafts

Table showing average healing times, in days from first graft, of different areas of full-thickness skin loss and the influence of size of grafted area, date of grafting, and use of penicillin.

Size of area	0–99 sq. cm. *Days*	100–299 sq. cm. *Days*	300–499 sq. cm. *Days*	500–999 sq. cm. *Days*	1000 sq. cm. and over *Days*
Effect of Grafting within 5 Weeks					
First graft within 5 weeks	10 (10 cases)	15 (32 cases)	23 (14 cases)	22 (13 cases)	21 (17 cases)
First graft after 5 weeks	19 (23 cases)	21 (36 cases)	38 (12 cases)	22 (8 cases)	34 (27 cases)*
Effect of Local Use of Penicillin-Sulphathiazole Powder					
With penicillin	16 (16 cases)	15 (39 cases)	25 (13 cases)	16 (12 cases)	30 (30 cases)*
Without penicillin	21 (17 cases)	20 (29 cases)	35 (13 cases)	29 (9 cases)	27 (14 cases)
Overall Average Figures					
All cases	18 (13 cases)	17 (68 cases)	29 (26 cases)	22 (21 cases)	29 (44 cases)

* These groups included 11 cases with full-thickness skin loss of 2000 sq. cm. and over.

Excluding patients who have lost 2500 sq. cm. of skin and more, the important thing in healing is not the size of the burnt area, but the resurfacing programme and the time-table to which it is geared.

The second principle of fundamental importance in healing raw areas is to graft early. We had long realized this as a clinical impression based on experience of particularly striking cases. Thus a phosphorus burn with loss of 1000 sq. cm. of thigh skin, whose sloughs were removed on the 18th day was grafted on the 21st day and completely healed on the 26th. A cylindrical burn of thigh and calf caused by petrol with destruction of 2500 sq. cm. of skin was admitted on the 15th day, had his sloughs cut off on the 18th day, was grafted on the 20th and 25th days, and was substantially healed by the 35th. In both these cases every patch of graft stuck and grew briskly. The consolidated figures for all cases grafted early confirm this impression; early grafts take best, grow more rapidly, and the raw areas are healed 1–2 weeks sooner.

The table also analyses the effects of penicillin on healing times. The 192 cases fell into two approximately equal groups; those treated without and those treated with local penicillin. The penicillin-treated groups consistently showed a shorter healing time. In assessing these results it is necessary to mention that the two groups were admitted for treatment at approximately the same times after

A

B

C

D

E

F

FIG. 357.—This series illustrates a case of burns of about 70 per cent of body surface. He had total skin destruction of almost the whole of both lower limbs and the whole of both buttocks.

Cause: Petrol accident. Site: Lower limbs, trunk, face, arms, forearms, hands. Percentage of body surface: 70. Area of full-thickness skin destruction: 5000 sq. cm.

A, Shows the appearance of the buttocks on admission at the sixth week. There is full-thickness destruction of all except the natal cleft.

B, Shows the appearance of the buttocks 3 days after grafting with dermatome-cut patches. The vaselined paper has been taken off some of the patches. Almost all have taken.

C, Shows the buttocks 3 months later, when he was completely healed, up, and active. The skin is durable and the patches have blended in well.

D, Taken after the second operation, shows the back of the left thigh with a dermatome-cut sheet graft stuck on to his popliteal fossa with gum acacia and patch grafts on the thigh, where suppleness is less important.

E, F, Show his appearance when healed. The donor sites on the trunk and arms, some of which have been used twice, can be seen. He had five grafting operations altogether.

His recovery was complicated by persistent diarrhœa and an attack of malaria.

being burnt, but that probably the grafting technique was better in the second group. The effect of penicillin has, of course, been to control infection and not as a direct epithelial stimulant.

Final Appearance.—The final appearance of a burn healed by patch grafts depends on three factors: thickness of the patch, the closeness of the packing, and the manner in which each particular patient heals.

Thin patches closely packed heal with a smooth, flat surface. In the early stages the appearance is chequered, but within 6–8 weeks the patchwork quality begins to fade and the striæ uniting the patches appear as a faint pink network on a *café au lait* background. The thicker, the bigger, and the more widely spaced are the patches, the more persistently unsightly is the final scarring. Thick big widely-spaced patches remain as slightly raised white plaques in areas of pink smooth epithelium formed by marginal outgrowth from them.

DEATHS

Table V shows the cause, percentage of body surface affected, chief depth of burn, area of full-thickness skin loss, primary treatment, day of admission and death, and the cause of death.

These nine deaths give a late mortality for a series of pre-eminently serious burns, in North Africa and Italy, of approximately 1 per cent.

This mortality contrasts with an early mortality of 5 per cent (7 deaths) in the 150 cases in this series whose treatment was undertaken within 24 hours of burning. Nearly 100 of the primary burns were from three naval incidents, and were caused by

A

B

C

D

FIG. 358.—This series illustrates a case of burns of about 45 per cent of body surface by a petrol cooker accident, which was healed by two patch-grafting operations with a 3-day interval during the eighth week.

Cause: Petrol accident. Site: Lower limbs, face, forearms, and hands. Percentage of body surface: 45. Area of full-thickness skin destruction: 3500 sq. cm.

A, Taken when he was first seen, shows granulating full-thickness skin destruction of about 75 per cent of both lower limbs; his face and hands and arms had healed at this stage. Bilateral tibial crest sequestra are seen and the left one is shown in B.

B, Sequestrum of crest of left tibia, removed at the first grafting operation, as it was found to be loose. Its bed was immediately covered with patch grafts, which all took. The right sequestrum was left, as it was firm, and it separated naturally a month later.

C, D, Show his appearance when healed and ambulatory, 5 weeks later, and show also the four dermatome-graft donor sites on his trunk.

This patient was grafted during an attack of acute glomerulonephritis, which started a month before grafting and persisted for a month after. During all this time he had hæmaturia, blood-urea of 50–70 mg./100 c.c. and blood-pressure about 170/110. After the second operation he had a uræmic fit, but his course was otherwise uneventful.

Table V.—THE 9 LATE DEATHS IN 800 CONSECUTIVE BURNS

No.	CAUSE	BODY AREA	CHIEF DEPTH	TREATMENT	DAY OF ADMISSION	DAY OF DEATH	CAUSE OF DEATH AND COMMENT
Part A. (The 3 deaths in 192 cases grafted).—							
1	Tank casualty	40 per cent	Full-thickness loss: 2000 sq. cm. (approx.)	S.V.G., Patch grafts	9	42	Friedländer pneumonia of blast lung
2	Tank casualty	40 per cent	Full-thickness loss: 2000 sq. cm. (approx.)	S.V.G., Patch grafts	27	40	Prolonged anæsthesia under 2·5 g. pentothal. Half raw area healed
3	Air crash	5–10 per cent	Epidermal: infective thickness loss	G.V. tan, 5th full day gross infection, late penicillin	38	49	*Staph. aureus* pyæmia
Part B (The 6 other late deaths in 800 consecutive serious burns).—							
4	Phosphorus	40 per cent	Full-thickness loss and dermal	G.V. tan, sulphathiazole	2	8	Agonal *Staph. aureus* in blood. ? early acute liver atrophy
5	Cordite	40 per cent	Epidermal, dermal	S.V.G., prophylactic and therapeutic penicillin	6	21	Prostatic and pulmonary abscesses. Burns dry and healed
6	Cordite	10–15 per cent	Epidermal	S.V.G., penicillin	5	11	Thyroid and pulmonary abscesses. Burns dry and healed
7	Tank casualty	30–35 per cent	2000–3000 sq. cm. F.T.L.	S.V.G.	8	8	Died a few hours after admission. Jaundice ++. Hb 10 per cent
8	Tank casualty	30–35 per cent	2000–3000 sq. cm. F.T.L.	S.V.G.	8	8	Died a few hours after admission
9	Cordite	40 per cent	Epidermal and dermal	S.V.G.	1	8	Adrenal hæmorrhage

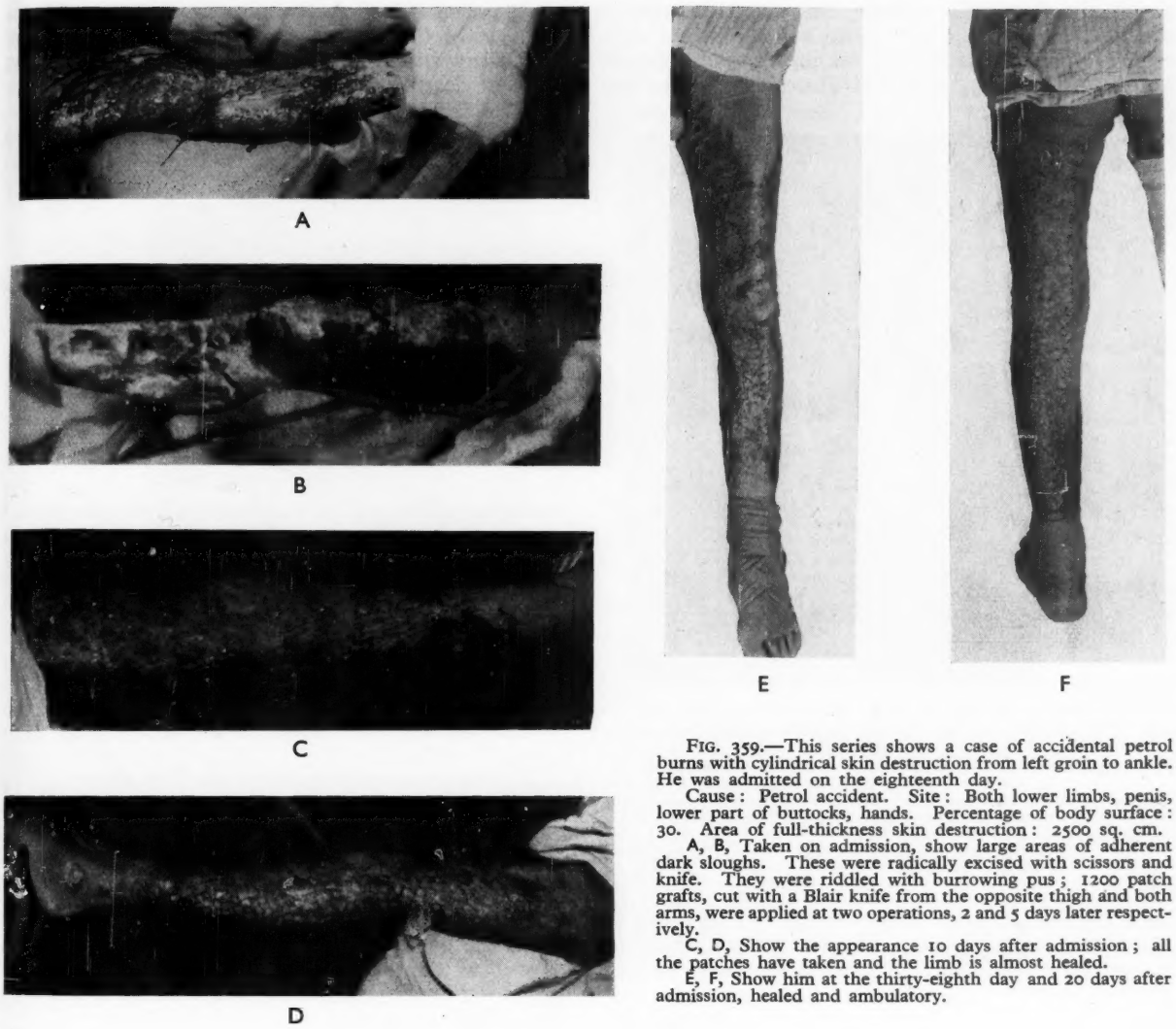

FIG. 359.—This series shows a case of accidental petrol burns with cylindrical skin destruction from left groin to ankle. He was admitted on the eighteenth day.

Cause: Petrol accident. Site: Both lower limbs, penis, lower part of buttocks, hands. Percentage of body surface: 30. Area of full-thickness skin destruction: 2500 sq. cm.

A, B, Taken on admission, show large areas of adherent dark sloughs. These were radically excised with scissors and knife. They were riddled with burrowing pus; 1200 patch grafts, cut with a Blair knife from the opposite thigh and both arms, were applied at two operations, 2 and 5 days later respectively.

C, D, Show the appearance 10 days after admission; all the patches have taken and the limb is almost healed.

E, F, Show him at the thirty-eighth day and 20 days after admission, healed and ambulatory.

phosphorus, cordite flash, and superheated steam respectively. These early deaths were in burns which involved 60 per cent, 70 per cent, 80 per cent (2), and 90 per cent (3) of the body surface.

In the cases grafted none of the deaths was primarily due to burns. One was a tank casualty with extensive blast effects in his lung who died of a Friedländer pneumonia on the 42nd day when he was practically healed. The second patient had successfully had half his 2000 sq. cm. of raw area resurfaced, and had by then regained normal sleep, appetite, pulse, and temperature. At his second operation, which lasted 2½ hours, 2·5 g. pentothal were used and he remained in deep coma with hyperthermia until death 24 hours after operation. Deep post-operative coma and hyperthermia were observed in other burns cases given 1 g. and more of pentothal during that summer.

The third death in the grafted series was due to sepsis. The patient died of staphylococcal pyæmia on the 11th day after admission, 49 days after sustaining epidermal burns of the hands, wrists, face, and neck. The burns had been coagulated with gentian violet on the 5th day. On admission on the 38th day, he was grossly emaciated, had multiple bedsores, and his burnt areas were gelatinous hypertrophic granulations with profuse *Staph. aureus* and hæmolytic streptococcal pus. His pyæmia and death were not inevitable results of his burns, which involved less than 10 per cent of the body surface and were initially superficial. It is thought that the pyæmia and fatal result were caused by late coagulation of an infected burn combined with inadequate nutrition.

On the basis of the cases described here, it is fair to suggest that death is not inevitable with burns up to 70 per cent body surface, with areas of full-thickness skin loss of up to 5000 sq. cm., which have thorough general treatment and prompt surgical resurfacing; but these severe deep burns are very prone to grave complicating conditions, which in this series included typhoid, glomerulonephritis, and malaria.

A man can be seriously ill for a week with only 10–20 per cent of his body surface burnt, but he should not die. All cases with 30 per cent of the body burnt are seriously ill for at least 2–3 weeks. When the area involved reaches 50–60 per cent of

the body the odds remain for a month against survival.

The four deaths from sepsis in the total series of 800 burns (Nos. 3, 4, 5, and 6) occurred in 10 per cent (2) and 40 per cent (2) burns. They were all staphylococcal pyæmias and must be considered to have been preventable. Two of these staphylococcal pyæmias had full penicillin, one late penicillin, and the fourth full sulphadiazole. The remaining three fatal burns (Nos. 7, 8, and 9) comprised one who had an adrenal hæmorrhage, and two in which 30–35 per cent body surface involved fat and muscle for most of their extent and who died a few hours after their admission on the 8th day. The exact cause of death in these cases is not known. Death was certainly accelerated by a 200-mile evacuation when the patients were critically ill; but the depth as well as the area of the burn is important in the prognosis for life, and these two cases were probably fatal from the start.

A B

FIG. 360.—This set illustrates a case of electric burns of trunk and scalp, involving the skull. His trunk was covered by patch grafts in the usual way. The area was infected with hæmolytic streptococci and is one where fixation of the grafts is often unsatisfactory. Three grafting operations were needed.

His scalp burns exposed a bare area of external table of the occipital bone. This was treated by removing the external table with a chisel and immediately applying patch grafts to the diploe. All these grafts took.

Cause: Electric. Site: Back of trunk and scalp. Percentage of body surface: 15. Area of full-thickness skin destruction: 1200 sq. cm.

A, Shows the burns of his back healed after grafting.

B, Shows the appearance of his scalp after healing of the patch grafts applied to the diploe exposed at operation.

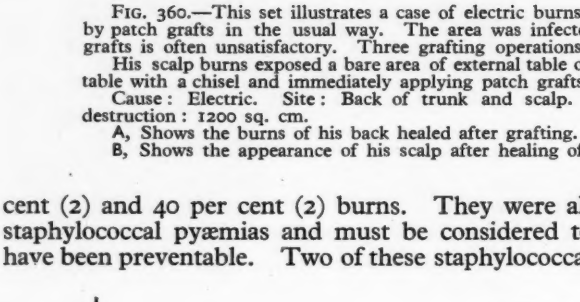

FIG. 361.—Graph showing relation of percentage of body surface involved plotted against mortality. The mortality is less than 1 per cent below 30 per cent body surface; 10 per cent between 30 per cent and 35 per cent; 20 per cent at 40 per cent body surface; 80 per cent above 60 per cent body surface; and when 80–90 per cent of the body surface was burnt all cases died.

SUMMARY

The management of 800 serious burns and the resurfacing problems of 192 of these during the healing and rehabilitation phases are described.

The history, recognition, and course of burns destroying the full thickness of the skin are detailed, and the reasons are given why it is important to assess the depth of burn early.

The preparation for grafting and methods of cutting and applying skin to extensive and to special areas are described, and the importance of carrying out a rapid resurfacing time-table for these cases is stressed.

The local complications of burns, infective, cicatricial, and dermatological, are discussed.

Results of 192 cases are given which show that all areas up to 2500 sq. cm. should be healed in 20–35 days from being burnt; areas of 2500–5000 sq. cm. have to be resurfaced at a slower rate and take 40–60 days.

The results recorded demonstrate the beneficial effects of early removal of sloughs, and the employment of penicillin, on the success of grafts and on healing times.

The early and late mortalities in 800 serious burns are given; and the causes of the 1 per cent late mortality in this series in discussed.

It is a pleasure to express our thanks to Major Roche and Captain Clynick for their expert anæsthesia, which was available for most of the cases; to Captain Grossmann, who treated many of the cases completely, and who had the ward care of most of them; to L/Cpl. Gale for his most expert dressings and assiduous care of patients in the saline bath: and the theatre staff—Corporal Cordery, Corporal Parker, and Private Farwell, who, under Sister P. Clutton, consistently provided theatre services of the highest standard by unremitting dreary work.

INJURIES TO THE PANCREAS AND THEIR SURGICAL TREATMENT*

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THE pancreas by virtue of its deep situation is well protected from the effects of the various injuries and insults which may be inflicted upon the abdomen and which form part of the hazards of everyday life. It is not surprising, therefore, to find that injuries to the pancreas are uncommon and few surgeons have an extensive experience in dealing with them. As might be expected, too, most injuries of the pancreas are associated with extensive injuries to other abdominal viscera, the liver, spleen, and stomach being most commonly involved, and are thus often beyond the reach of surgical aid. However, the close apposition of the body of the pancreas to the unyielding body of the first lumbar vertebra makes it possible for the pancreas to be crushed or ruptured or torn across in crush injuries of the upper abdomen, and the more so if the trauma is a very sudden one which produces its effects before the abdominal musculature has time to guard against the blow. In such cases the pancreas may be injured apart from any other viscus and Garré in 1905 was able to find 8 cases in the literature in which the pancreas was the only organ to be injured, including one personal case of rupture of the pancreas which he was able to cure by suturing. Most collected series of cases of pancreatic injury have included a number in which the injury has been produced by bullets and other missiles or by knife or bayonet, but it is not the purpose of this paper to consider wounds of the pancreas which may result from warfare, but rather those resulting as suggested above, from crush injuries of the abdomen. The trauma which may give rise to such injuries may be surprisingly slight and is often not sufficient to produce any wounding or bruising of the parietes; and this is probably a reflection of the flaccid condition of the abdominal musculature at the time of the accident which in itself made the injury possible. Such an injury may vary from complete rupture of the gland to simple bruising, and naturally the condition of the patient will vary accordingly. In the severe injuries with complete rupture there will be no doubt in the mind of the surgeon that laparotomy will be required, although often no exact preoperative diagnosis can be made. In this connexion attention may be drawn to the importance of left shoulder-tip pain in injuries to the pancreas which produce effusions into the lesser sac; a case in which this formed an important feature was reported by A. L. d'Abreu in 1935, and is among the series of cases reported later in this paper.

There remains, however, a number of cases in which no lesion of the pancreas is suspected at the time and in which the condition of the patients is such that they escape immediate laparotomy only to return at a later date with symptoms which are directly referable to the original injury. The following are given as typical case histories of such patients, who form a most fascinating and important group of pancreatic injuries:—

Case 1.—L. H., aged 16, while at work on Monday morning, Jan. 12, 1941, in the colliery, was knocked down by a runaway coal truck which struck him in the upper abdomen on the left side. He experienced great pain at the time and was sent home by ambulance and put to bed. He was seen by his doctor, who kept him in bed and ordered Epsom salts. He vomited greenish material on Monday and Tuesday and the pain in his abdomen remained, being aggravated by deep breathing. On Thursday he was admitted to the Cardiff Royal Infirmary as an emergency under the care of the Surgical Unit. On examination he was found to be pale; pulse 102; T. 100°. He was complaining of upper abdominal pain. There was a slight abrasion on the abdomen above and to the left of the umbilicus, tenderness and some muscle guarding in the left hypochondrium. Urine was normal. He rapidly improved on expectant treatment and was discharged completely symptom-free on Jan. 23, after 11 days in hospital.

He was re-admitted again on Feb. 11, 1941. He said that since returning home he had had two attacks of upper abdominal pain associated with vomiting, each lasting several days, and that a month after his original injury he had noticed for the first time a swelling in the upper abdomen, for which his doctor had advised his re-admission. On examination, he was found to have a tense swelling in the left hypochondrium, the outline of which is shown in the photograph (*Fig.* 362). A barium meal showed a large filling defect in the stomach consistent with an extragastric mass. Laparotomy was carried out on Feb. 18, 1941, and a pseudo-pancreatic cyst found presenting between the stomach and colon. It was found to be too inaccessible for easy marsupialization and was accordingly aspirated and the abdomen closed with a drainage tube down to the site of aspiration. The wound healed without drainage and the patient was discharged on March 17, 1941.

He was re-admitted for the third time on Oct. 16, 1941; gave a history of attacks of abdominal pain which

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* Being a Hunterian Lecture delivered at the Royal College of Surgeons, May 4, 1945.

doubled him up and which occurred about once a fortnight, lasting a few hours. On examination, he was found to have a large swelling in the left hypochondrium, rather bigger than at the previous admission. Laparotomy performed Oct. 30, 1941; cyst the size of a football, 2½ pints of cloudy fluid aspirated; marsupialized and drained. Discharged Nov. 22, 1941—healed.

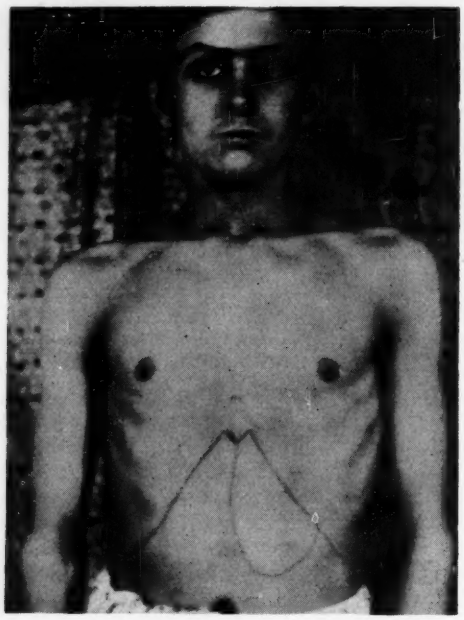

FIG. 362.—Photograph showing outline of swelling in left hypochondrium.

He has been admitted once since, in 1943, under the care of the physicians with catarrhal jaundice—otherwise he has remained well.

Case 2.—G. H. C., aged 37, was admitted to Llandough Hospital on Nov. 5, 1942, under the care of Mr. D. J. Harries, having been thrown out of a lorry and sustained injuries to chest and abdomen. Pain and tenderness were present in left hypochondrium, but no rigidity. Several attacks of pain and vomiting and abdominal distension while in hospital. Discharged from hospital Dec. 21. Re-admitted Feb. 10, 1943, with a large mass in the upper abdomen, which was found on laparotomy to be a large pseudo-pancreatic cyst. It was marsupialized and drained.

These cases will suffice to illustrate the way in which trauma may produce late results in the form of a cyst or pseudo-cyst of the pancreas. It must not be supposed that such conditions are common; indeed, a careful search through the Cardiff Royal Infirmary records for the last fifteen years reveals only 2 cases out of about 75,000 total in-patients treated in the Infirmary during the same period, and this accords with the experience of others. Judd, Mattson, and Mahorner (1931), in one of the largest series of collected cases of pancreatic cyst, report on 88 cases from the Mayo Clinic out of 723,397 surgical patients admitted during the same period. When it is remembered that less than 25 per cent of these 88 were cysts due to trauma, the incidence of the condition is seen to be 1 in 40,000 and its rarity will be appreciated.

It is very noticeable in all the reported series that cysts following on inflammatory conditions of the pancreas are very much commoner in women than in men, being commonly associated with chronic cholecystitis and cholelithiasis, while those resulting from trauma occur more commonly in males, in association with the greater hazards of their occupations; all 3 cases in this paper were in males. The time interval between the infliction of the trauma and the appearance of the cyst is very variable, from a few days to over a year, but many cases seem to present within a month or six weeks of the injury, which may be taken as typical. The pathological processes which lead to the formation of a cyst in such typical cases would appear to be as follows: Bruising of the pancreas produces a hæmatoma beneath the capsule. Damaged pancreatic cells release their ferments into this; fat necrosis is often seen in immediate operations upon pancreatic injuries. The trypsin in the juice, while normally inactive as secreted, becomes activated by contact with damaged and devitalized tissues and digests the clot, thus giving rise to further bleeding from the thin-walled pancreatic vessels. This process is repeated and the cyst enlarges as a result of repeated hæmorrhages. The digestion may go on until the pancreatic duct is involved, when the size of the cyst will be rapidly increased by the admixture of pancreatic juice. No doubt there are many variations of this process and the cysts of rapid onset are probably due to rupture of the capsule of the pancreas, frank hæmorrhage, and escape of pancreatic fluid into the lesser sac of the peritoneum. However, the above seems to be the ordinary mechanism, and the growth of the cyst by means of repeated hæmorrhages is stressed, as it may well form a basis for the earlier recognition of the condition. It would appear that the critical period for the recurrence of hæmorrhage into a cyst is from 10 to 14 days after injury, and from a careful examination of the case records it is evident that the hæmorrhage when it occurs frequently produces an attack of epigastric pain and vomiting in the patient, associated with varying degrees of shock. Both the case histories quoted give evidence of this. The occurrence of such attacks, therefore, in a patient who appears otherwise to be recovering satisfactorily from upper abdominal trauma should lead the surgeon to suspect the onset of a pseudo-pancreatic cyst.

In the established case there should seldom be any difficulty in making the diagnosis, as the characteristic swelling in the left hypochondrium may be easily seen as well as felt.

Pancreatic cysts may extend in various directions, above the stomach, between the stomach and the colon, or downwards behind the colon into the pelvis, but by far the commonest is between the stomach and colon, when it will produce the classical appearance of swelling in the epigastrium or left hypochondrium.

Of the accessory methods which we have for confirming the clinical diagnosis, the most consistently valuable is the barium meal radiographic examination, when a variety of deformations of the stomach or duodenum may be shown produced by the pressure of the large extragastric mass. Diagnosis having been established, it remains to consider the appropriate surgical treatment. In 1862 Le Dentu punctured a cyst of the pancreas and drained it under

the impression that it was a cyst of the liver. When the patient died of peritonitis he somewhat gloomily wrote :—

"Cysts of the pancreas should be relegated to the list of those affections where the healing art is impotent."

While we should agree with him in his condemnation of needling either as a method of diagnosis or treatment, surgery has advanced since his day and now holds out three possible and satisfactory forms of treatment for the condition. The three types of operation which have been advocated and applied are excision, marsupialization and drainage, and primary anastomosis between the cyst and the stomach or small intestine. Of the first it may be said that, while the most radical, it is seldom practicable for technical reasons. The second method has stood the test of time and experience and probably represents the standard procedure at the present day. It has the merits of being easy and of universal application and in fully 80 per cent of cases it produces a rapid cure of the condition. Against this may be set the fact that recurrences are by no means unknown, and in some cases where there is major damage to the pancreatic duct or ducts the drainage of the cyst may be followed by the development of a troublesome and intractable pancreatic fistula, although in most cases the fistula will close spontaneously in course of time. The third method is of more recent origin and has been introduced with a view to overcoming the objections which have been advanced above to the method of treatment by marsupialization and drainage. It has not yet got the weight of experience behind it that marsupialization and drainage have, but so far as it has been tried it is probably the most satisfactory line of treatment which we have to date. Of the 7 cases reported in this paper 5 were treated by marsupialization, 1 by evacuation of the cyst and closure of the abdomen, and 1 by anastomosis with the stomach over a rubber tube. No more will be said at this stage as it is our purpose to return to a further discussion of this point later in the paper. *Table I* gives a summary of the 7 cases.

It will be noticed that in one of these cases drainage was followed by the development of a pancreatic fistula, which leads me on to this further sequel to pancreatic trauma which may confront the surgeon, and which has its own peculiar and fascinating problems.

Most of the pancreatic fistulæ which have been recorded in the literature have followed the drainage of pancreatic cysts and it may be inferred that if pancreatic cysts are of rare occurrence, pancreatic fistulæ will be even rarer, and this is borne out in fact. However, pancreatic fistulæ may follow on immediate operations for pancreatic trauma and from other operations in which the pancreas may be damaged; notably gastrectomy, and, of course, from any operation upon the pancreas itself. It is noticeable in this connexion that now that extensive operations upon the pancreas are coming within the field of surgery the number of pancreatic fistulæ being reported in the literature is steadily rising, and this in itself would be adequate reason for discussing so rare a condition, for it may be that in the future it may become a far more common complication of surgery than it is at present. For myself I can say that the study of the one case which I have had under my care has proved most stimulating and has led me into the byways of physiology and biochemistry which are unfamiliar haunts for the surgeon and which have provided a welcome excursion for the mind which in these days of specialization too easily resigns itself to the familiar scene and the well-trodden path. It will perhaps be appropriate at this stage to recount the story of the case of pancreatic fistula which it has been my fortunate lot to observe and to pass from this to a discussion of the various problems which these cases present, and the treatment appropriate to the condition.

Table I.—Report on 7 Cases of Pseudo-pancreatic Cyst

Case	Age	Sex	Aetiology	Duration of Onset	Treatment	Result
L. S.	16	M.	Crush injury to abdomen	1 month	Aspiration-recurrence. Marsupialization and drainage	Cured
L. L.	50	F.	Subacute pancreatitis	1 month	Cyst anastomosed with stomach over a tube	Cured
G. H. C.	37	M.	Blow on abdomen	3 months	Marsupialization and drainage	Died
F. B.	57	M.	? Carcinoma of pancreas	18 months	Marsupialization and drainage. Implantation of resulting fistula into stomach, 5 months later	Died 1 year from generalized carcinomatosis of undetermined origin
C. D.	69	M.	Acute pancreatitis	5 days	Marsupialization and drainage	Cured
B. G.	7	F.	Acute pancreatitis	5 days	Evacuation of large blood-clot in lesser sac, abdomen closed	Cured
A. W.	13	M.	Blow on abdomen	1 month	Marsupialization and drainage	Cured

Case 3.—On the afternoon of Feb. 24, 1944, there was admitted under my care at the Whitchurch Emergency Hospital a small boy aged 9. His story was that shortly after lunch that day, whilst engaged with helping with the milk round, he had been unfortunate enough to meet with an accident. He had been sitting on the back of the van, which started suddenly, throwing him off on to the ground, and which then added insult to injury by backing, so that one of the rear wheels ran over his upper abdomen. The child had vomited almost immediately and had been taken home in the van, vomiting again on his arrival. He was seen by his doctor,

who sent him straight into hospital, where he arrived two hours after the accident.

When I saw him the same afternoon he was sitting up in bed calmly studying a coloured comic and he did not seem much distressed and was able to give a clear account of the accident. His temperature was 97° and his pulse 80. There was a slight abrasion on the abdomen just above the left anterior superior spine, but there was little tenderness and no rigidity. He was accordingly treated conservatively and put on a half-hourly pulse record and food and fluids withheld. His pulse rose slowly during the night and was 104 when seen in the morning, when he vomited again, the vomit being projectile in character, and was observed to have visible peristalsis in the region of the umbilicus. Laparotomy was, therefore, carried out immediately.

On opening the peritoneal cavity there was a considerable escape of dark blood and it was immediately seen that there was a rupture of the liver extending from the inferior edge down into the portal fissure and almost completely avulsing the quadrate lobe, which was accordingly removed. The lesser omentum was torn and there was also seen to be bruising and hæmorrhage into the body of the pancreas and several well-marked areas of fat necrosis in the neighbourhood. A routine exploration of the intestinal tract produced the surprising finding of an entero-enteric intussusception about 6 in. long at about the mid-point of the small intestine. This was easily reduced and no cause for it was seen or felt. There was no other injury in the abdomen. The rent in the liver was sutured and the abdomen closed with a rubber drainage tube down to the damaged pancreas.

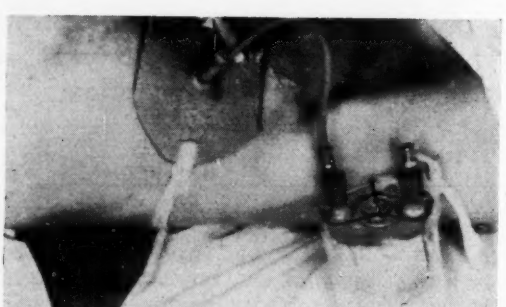

FIG. 363.—Drainage of fistula through a catheter leading into a bottle.

The drainage tube was removed on the second day, but on the fourth day a clear, watery discharge appeared at the site of the tube and rapidly increased in quantity until the dressings were constantly wet through. A specimen of the fluid showed it to be pancreatic juice with a *p*H of 9 and the diastase estimation gave a value of 2000 Wohlgemuth units. Some superficial excoriation of the abdominal wall appeared in the next few days, but at no time was there any serious digestion of the abdominal wall and even the slight excoriation which was present was extremely ephemeral, sometimes being present in the morning, but completely healed by the same evening. Within a few days it was possible to insert a catheter into the fistula and drain the fluid into a small bottle (*Fig.* 363). Following the development of the fistula the boy's condition steadily deteriorated. He rapidly developed the features of extreme dehydration and this was aggravated by attacks of vomiting, which were often projectile and violent in character. His fluid and salt balance was maintained by rectal salines to supplement the fluid by mouth. Loss of weight was progressive and severe and this was accompanied by anorexia and marked asthenia and he was soon reduced to little more than a living skeleton. Curious mental changes also appeared at this time manifested by several attacks in which the patient would suddenly rouse himself from his weak, almost stuporose condition, to become transformed momentarily into a wild animal at bay. In these attacks the boy would sit up in bed with bared teeth, and convulsively clenched hands, ready to bite or scratch anyone who approached, and emitting, from time to time, wild and piercing screams. He would pass rapidly from this state back into his stuporose condition and had no memory of the attacks afterwards.

About a fortnight after the onset of the fistula the patient, who had been better for two days, slipped into the semi-comatose condition which has been so well described by Whipple and others under the term "pancreatic asthenia". In this condition the pulse was almost imperceptible and the breathing so shallow that the boy had every appearance of death. An intravenous saline was at once started, and within a very few minutes the boy revived. From this time progress was slow but steady, although the extreme wasting remained for some time. The fistula closed spontaneously in two months, and following this the patient rapidly put on weight and was discharged from hospital on May 25. He has remained well since this time.

The above clinical record raises many problems in connexion with pancreatic fistulæ which have engaged the attention of a number of investigators and which may be summarized as follows :—

1. The volume and control of pancreatic secretion.
2. The cause of death in fatal cases and of pancreatic asthenia in less severe ones.
3. The degree of importance of the external secretion of the pancreas to normal digestion.
4. The enzymatic activity of pancreatic secretion.
5. The treatment of pancreatic fistulæ.

1. The Volume and Control of Pancreatic Secretion.—It is generally acceded that the pancreatic secretion is under the control of both nervous and humoral influences. The humoral control is effected by means of secretin which is liberated from the duodenum in response to the discharge of the acid contents of the stomach into it. The secretin thus thrown into the blood-stream stimulates the pancreas to produce a large volume of secretion rich in sodium bicarbonate. Stimulation of the vagus on the other hand, has a much smaller effect on the volume secreted, but produces a fluid which is rich in digestive ferments. Thus it will be seen that the volume and alkalinity of the secretion are largely under humoral control while the digestive activity is under nervous control. The secretin mechanism contains within it the elements of an automatic regulator or cut out, for the alkaline secretion poured out in response to the acid chyme entering the duodenum rapidly brings about its neutralization and consequently the cessation of the stimulus. It is quite clear that in cases of fistula this automatic control is rendered inoperative. The pancreas responds to the secretin stimulus, but the resultant flow of fluid does not reach the duodenum. It is for this reason that I believe that all inferences as to the normal output of external pancreatic secretion, based upon data derived from cases of fistula, are probably erroneous and tend to be much too large. Probably they represent the maximum output of the gland, working at full pressure in response to

over-intense and over-prolonged secretin stimulation. However this may be, the volume of fluid produced in fistula cases is certainly very impressive and values up to 2000 c.c. have been recorded; the

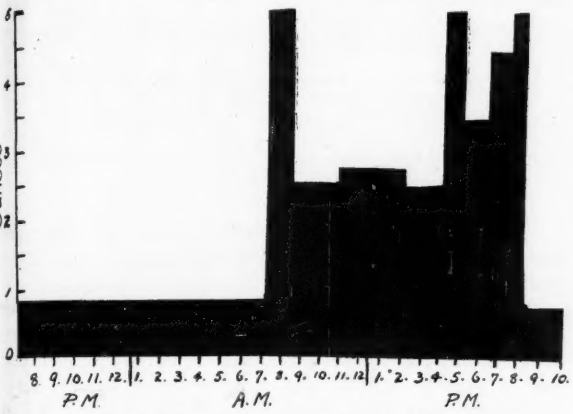

FIG. 364.—Record of output of pancreatic fistula for 24 hours. The increase in secretion in response to food is well shown.

case I have reported produced as much as 46 oz. in the 24 hours (*Fig.* 364). The secretion in pancreatic fistulæ is also continuous, going on by night and day, but again I think it would be wrong to assume that this is necessarily the case in the normal individual for the reason given above. In this connexion attention may be drawn to the conflicting results obtained by vagal stimulation and vagotonic drugs. Many observers have reported a drop in the volume of secretion following such measures. Apart from the fact that such vagatonic drugs usually stimulate gastric motility and hydrochloric acid output, and hence might be expected secondarily to produce an increase in volume of pancreatic secretion via the secretin mechanism, the result should not surprise us. What is more significant is the effect of such drugs as pilocarpine, etc., on the enzymatic activity of the fluid, and little or no work in the human has been done on this problem. The absence of evidence for psychic secretion reported by several workers is also to my mind inconclusive, as the only index of secretion commonly used is the volume.

2. The Cause of Death in Fatal Cases and of Pancreatic Asthenia in Less Severe Cases.—It was shown experimentally by McCaughan (1929) more than 15 years ago that complete pancreatic fistulæ in dogs were invariably fatal in from 5-8 days. If death did not occur in this time after the experimental establishment of a fistula it was invariably found at autopsy that the fistula was not complete and that some part of the pancreatic secretion was finding its way into the duodenum. The cause of death in fatal cases was thought to be mainly the profound anhydræmia resulting from the fistula. The blood-chemistry was found to be little altered apart from a slight reduction in the chlorides and a terminal rise in non-protein nitrogen. There was no constant variation in the carbon-dioxide-combining power of the plasma. They further established that intravenous administration of saline prolonged life and maintained normal blood-chemistry and that administration of whole pancreatic juice by mouth restored even moribund animals to health. In dogs in which only a partial fistula was induced there was no constant clinical picture produced; some of the dogs suffering from dehydration and loss of weight and others remaining in comparatively good health. All this conforms fairly closely to the findings in human cases of pancreatic fistula. It is rare for a human pancreatic fistula to be complete and thus fortunately its fatal effects are seldom observed, but there is no doubt that they will occur and would have done so in the case cited apart from the institution of vigorous treatment. However, the cause of death in such cases is not yet sufficiently clear and is almost certainly due to a number of contributory causes. The dehydration is an obvious factor. Human pancreatic fistulæ may secrete up to 2000 c.c. (3½ pints) of fluid in a day, which represents a very substantial fluid loss. In addition to this there is the fluid lost in the vomiting which is such a common concomitant of the condition. This dehydration is rapidly reflected in the sunken eyes and inelastic skin of the sufferer. Then, too, pancreatic fluid contains an appreciable quantity of protein, albumin and globulin, and this mounts up in the large volume secreted to produce a serious drain on the plasma proteins. This is to some extent compensated for by the anhydræmia and reduction of blood-volume, so that the reading obtained may not be low unless it is interpreted in relation to the concentration of the blood (*Table II*).

Table II.—Case 3. REPORT ON BLOOD EXAMINATION (APRIL 10, 1944)

Titration for non-ionized alkali in the serum, gives similar results to those of normal blood.	
Total proteins	8·25 g. per cent
Albumin	6·0 g. „ „
Globulin	2·25 g. „ „

Pancreatic fluid also is a markedly alkaline secretion—the *p*H of the specimen recorded was 9; this alkalinity is due to the high sodium bicarbonate content, and it might be thought that such a constant drain in alkali would produce a significant acidosis. While there is no doubt that it does to some extent do so, the effect should not be over-estimated. In the first place it is to some extent balanced by the frequent concomitant vomiting, and also there is good evidence to show that much of the bicarbonate is produced locally in the pancreas as a product of the metabolism of its cells and is not derived from the blood. This is in keeping with the surprisingly slight changes in blood-chemistry which have been repeatedly reported even in patients who were gravely ill. The finding has been succinctly and wittily summarized by Capt. J. M. Miller and Major T. B. Wiper (1944) in a recent excellent paper. When reporting a serious case under their care they state: "He was in vibrant health in the laboratory, but practically dead in the ward."

However, although the blood-chemistry changes are slight and may be masked by the concentration of the blood, that is not to say that such changes which are present are not very important. Indeed, the writers referred to above draw attention to the similarity between the condition of pancreatic asthenia and the more extreme forms of Addison's disease and other states in which the blood-sodium is significantly lowered. They noted a fall in plasma-sodium in one of their cases from a normal of 142 m. Eq/L to 113·3 m. Eq/L, which is well below the

level regarded as diagnostic of Addison's disease. They also point out that reduction of sodium concentration is accompanied by a secondary loss of water, thus increasing the dehydration and enhancing the hæmoconcentration, elevation of blood-proteins, and finally non-protein nitrogen. The resemblance between Addison's disease and the victim of a severe pancreatic fistula is certainly striking, and it is probable that the sodium loss is an important factor in the production of the symptom complex. This discussion of blood-chemistry may seem to be unduly academic, but at least it has an important practical significance as it emphasizes the importance of maintaining the fluid and sodium reserves of the body in all such cases. The dramatic effect of intravenous saline in the patient I have described had to be seen to be appreciated. It is unnecessary to give sodium bicarbonate, as the body is able to supply adequate quantities of the bicarbonate ion as a by-product of its metabolic activities, but it could well be administered as an alternative to saline if vomiting is not present and there is a risk of chloride retention and resultant œdema. The value of whole pancreatic fluid by mouth could not be tested in my case as the patient was not sufficiently co-operative, but many workers have reported very favourably upon the effect of its administration.

While there is no doubt that the main factors contributing to the mortality and morbidity of pancreatic fistulæ are the anhydræmia and blood changes outlined above, there remains an uneasy feeling that this is not perhaps the whole story. It is difficult to understand, for example, why cases with a partial fistula should so often show little or no impairment of health even though the total fluid loss may approach or even surpass that from a total fistula. It raises in one's mind inevitably the question as to whether the external secretion of the pancreas does not supply some factor vital to the well-being of the body. The answer would appear to be conclusively in the negative, for it is known that total extirpation of the pancreas in man is not incompatible with life and even with health and may not even be accompanied by any disorder in the digestive processes. Possibly there may be some individual variation in this and clearly more work is needed on human pancreatic physiology to establish the point.

3. The Degree of Importance of the External Secretions of the Pancreas to Normal Digestion.—It has just been mentioned that the pancreas has been successfully extirpated in the human without impairment of digestion, and it may well be asked whether the external secretion is of any vital importance in the digestive processes. The occurrence of fatty diarrhœa in cases of pancreatic fistula and other pancreatic diseases is quite inconstant and apparently quite capricious, being unrelated to the severity of the condition (some cases of partial fistula have severe steatorrhœa, while other cases of total fistula have no such symptoms). Probably in most people the succus entericus together with the bile is quite capable of carrying on the digestive processes to completion if the pancreatic juice is wanting, and we have but another example of the prodigality with which the body is supplied with alternative mechanisms to take the place of any which may be temporarily or permanently put out of action. No doubt, however, there are some individuals who are not thus able to compensate for the loss of pancreatic secretion, and in these the withdrawal of this fluid represents an irreparable loss resulting in serious digestive impairment (*Table III*).

Table III.—Case 3. REPORT ON FÆCAL FAT

March 25, 1944
Total Fat: 24 per cent by weight of dried fæces
55 per cent of the total fat is split
45 „ „ „ „ „ „ „ unsplit

April 10, 1944
Total Fat: 6 per cent by weight of dried fæces
67 per cent of the fat is split
33 „ „ „ „ „ „ unsplit

This leads me to the fourth question concerning pancreatic fistulæ, viz., the enzymatic activity of pancreatic fluid.

4. The Enzymatic Activity of Pancreatic Secretion.—It is well known that the pancreas secretes three ferments; diastase or amylase, lipase, and trypsin, which act respectively upon carbohydrates, fat, and proteins. With regard to the first, it is constantly present in pancreatic juice, and indeed can hardly be said to be diagnostic of pancreatic fluid as it occurs in fair quantity, for example, in ascitic fluid. Lipase has also been reported present in most of the cases of pancreatic cyst fluid and fistula fluid in which it has been sought. The quantity of these two enzymes present is very variable from case to case and from day to day, e.g., in the personal case the diastatic index was 2000 Wohlgemuth units one day and only 200 units a few days later.

The main interest, however, is focused on the trypsin and the proteolytic activity of pancreatic juice. It must surely be a matter of comment in studying the literature concerning pancreatic fistulæ to find how variable have been people's experiences with regard to the proteolytic activity of fistula juice as evidenced by its tendency to produce digestion of the tissues of the abdominal wall. In some of the reported cases this has been a major complication, while others have remarked on its complete absence. In my own case it was never a problem and comment has already been made on its ephemeral character. The traditional explanation of these facts is, of course, that trypsin is secreted by the pancreas in an inactive form which is activated by a specific enzyme, enterokinase, in the duodenum; and in clinical support of this is adduced the fact that duodenal fistulæ are always accompanied by severe digestion of the abdominal wall. This would, of course, account for the absence of digestion in cases of pancreatic fistulæ, but on the other hand would not explain why some pancreatic fistulæ are accompanied by pronounced proteolytic activity. In order to account for this fact it must be presumed that other things besides enterokinase can bring about the activation of trypsinogen, or trypsin plus protein inhibitor as it is now generally believed to be. Many things have been claimed to be effective in such activation and among the list are calcium chloride, exposure to air, the presence of bacteria, bile, and bile-salts, etc. It is possible that all these may be effective at various times in splitting up the trypsin inhibitor complex, but that they are not always effective is shown in our own case. In one specimen which was kept for 24 hours prior to the assay

and which was found by culture to contain *Str. pyogenes*, *Str. viridans*, and *Ps. pyocyaneus*, no direct tryptic activity could be found, but it was present after activation by calcium chloride (*Table IV*).

Table IV.—Ronald Cokeley. ANALYSIS OF PANCREATIC FISTULA FLUID (MAY, 1944)

Full investigation of Pancreatic Fluid

Pancreatic juice:
- Total solids, 1·35 per cent
- Inorganic ash, 0·67 per cent
- Organic matter, 0·68 per cent (by difference)
- Calcium, 2·2 mg. per cent

Enzymes:
- Diastase—200 Wolgemuth units/c.c.
- Lipase, B.P. assay—8 c.c. N/1 per cent
- Trypsin, Martindale assay: No activity.
- Activated with 1/10 vol. 4·5 per cent $CaCl_2$ = 2·7 c.c. N/1 per cent
- Trypsin inhibitor—100 mg. per cent trypsin neutralized.

Bacteriology:
- *Str. viridans, Str. pyogenes, Ps. pyocyaneus*

The action of calcium chloride in this respect deserves comment, I think, in view of the fact that the same pancreatic juice was found to contain 2·2 mg. of calcium, a figure which corresponds closely with that cited by other authors including the recent investigation by Capt. J. M. Miller and Major T. B. Wiper. These authors draw particular attention to the calcium content of pancreatic juice as constituting a little recognized phase of calcium metabolism in which diffusible calcium from the blood-stream is constantly being discharged into the duodenum. They make the point that this must be ionizable calcium as only this fraction of the calcium of the blood is able to diffuse into extracellular fluid. However, it would not seem that this is necessarily the case, and the very fact that it does not produce activation of the trypsin is in favour of its being protein bound. The finding that bile-salts are effective in activating trypsinogen is, I think, most suggestive, and indeed it makes one wonder whether this is not the normal mechanism of activation. Intermittent regurgitation of bile into a pancreatic fistula would in this case account for the presence or absence of tryptic activity in fistula fluid from day to day. That bile-salts do, in fact, activate the trypsinogen of normal blood-plasma has been confirmed for me by Miss B. Still, to whom I am indebted for all the biochemical work on the case presented. Once again this may seem to be an academic discussion unrelated to surgical practice, but a clearer understanding of the mechanism involved in trypsinogen activation may well prove a help in dealing with other diseases of the pancreas.

5. The Treatment of Pancreatic Fistulæ.—We turn now, finally, to the treatment of pancreatic fistulæ. It should be said at the outset that fully 80 per cent of such fistulæ close spontaneously in due course, and such closure has been noted even up to a year after the fistula has been established. Treatment in the first instance should, therefore, be conservative and should be directed towards: (1) The restoration and maintenance of fluid requirements and normal blood-chemistry. (2) The protection of the wound when digestive activity is a feature of the condition. (3) Diminution in the volume of the secretion to encourage closure. The first of these has already been adequately dealt with. The second, as has been mentioned, is often no problem at all, but if digestion of the wound is troublesome probably the best method of treatment is that devised by Caryl Potter (1929) for the treatment of duodenal fistulæ. This is based on the fact that trypsin is only active in an alkaline medium, and consists in irrigating the wound with a continuous drip of N/10 hydrochloride acid and dressing it with gauze soaked in 10 per cent Witte's peptone to absorb any tryptic activity which may still be left. Less severe cases can be satisfactorily handled by protection of the skin by liberal applications of vaseline or aluminium paste.

The third objective of conservative treatment—namely, to diminish secretion in order to favour closure of the fistula—may be conveniently discussed under the headings: (*a*) Diet; (*b*) Drugs; (*c*) Other measures. Since the volume of the secretion is largely governed by the secretin mechanism, which is in turn dependent upon the motility of the stomach and the acidity of its contents, any measures which will lessen these might be expected to bring about a reduction in the volume of secretion.

a. Diet.—As long ago as 1907, as a result of extensive investigations, Wohlgemuth instituted his dietary regimen for pancreatic fistulæ and this has remained the standard since his day. His method was briefly to give a high-fat, low-carbohydrate diet together with plentiful doses of sodium bicarbonate and atropine; all of which measures are designed to diminish gastric acidity and motility. Many have reported favourably on this régime, but more recent and accurate measurements in the human have cast some doubt on its effectiveness. Accurate observations on the effect of various diets and drugs on the secretion of human pancreatic fistulæ have been made recently by McCaughan and Purcell (1941) and others, and these have demonstrated that a high-fat meal does not show up particularly favourably in comparison with other diets in effect on the volume of pancreatic secretion. Sodium bicarbonate by mouth in the usual doses also has but little effect. It must be remembered that the effect of sodium bicarbonate upon the stomach is to stimulate the production of more acid, and probably very massive doses are required to overcome this effect. Probably other alkalis such as aluminium hydroxide would have a more beneficial effect, but they do not seem to have been tried. It is quite certain that sodium bicarbonate direct into the duodenum through a duodenal tube markedly decreases the volume of secretion and that dilute hydrochloric acid equally dramatically increases it.

b. Drugs.—With regard to drugs, it would be tedious to enumerate the very large number of drugs which have been tested experimentally. Suffice it to say that ephedrine appears to be the most satisfactory for clinical use. There are other drugs, as for example physostigmin, which have a greater effect in diminishing pancreatic secretion, but they all have unpleasant side reactions of one sort or another which make them undesirable for clinical use.

c. Other Measures.—Some writers have reported favourably on the effect of deep X-ray therapy in diminishing pancreatic secretion and producing closure of fistulæ, but the consensus of opinion amongst most recent writers who have tried it is that it has little or no effect, and the same may be said for the use of sclerosing fluids injected into the

fistula. One case reported here had a trial of sodium morrhuate injections without effect. If the fistula does not close within a reasonable period, even with the use of the above measures, surgical treatment should be undertaken. In my judgement surgery should not be considered for at least six months if the patient's health is not being impaired by the fistula, as many fistulæ have closed after several months. If surgery is deemed advisable the fistula may be dissected out and implanted into an adjacent viscus; the stomach, gall-bladder, and jejunum have all been used for this purpose. Of these the stomach has found most favour and has much to commend it because of its accessibility and the fact that the tryptic activity of the juice which might digest the suture line is immediately destroyed by contact with the acid secretion of the stomach. No untoward effects upon the digestive processes have been observed. However, "prevention is better than cure" in surgery as in medicine, and the fact that most pancreatic fistulæ follow upon the drainage of pancreatic cysts must, I think, compel the question as to whether primary anastomosis of pancreatic cysts should not replace marsupialization and drainage as the standard treatment for pancreatic cysts.

Naturally the suggestion must be treated with reserve and judged primarily upon the mortality figures. Unfortunately, the method has not been sufficiently frequently applied to give reliable data in this respect, but so far as the method has been used it has given a good account of itself and compares very favourably with marsupialization, which carries an appreciable mortality. Lahey and Lium (1937) collected 8 cases of primary anastomosis as a treatment for pancreatic cyst, without any mortality and with uniformly good results; one of these cases is included in this present review. Clearly the method is deserving of an extended trial and may well prove to be the treatment of choice in the future.

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PAROXYSMAL HYPERINSULINISM DUE TO ISLET-CELL TUMOUR OF THE PANCREAS

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ENDOGENOUS hypoglycæmia was first recognized as a clinical syndrome by Seale Harris in 1924, following the recognition of the effects of overdosage of insulin.

In 1927 Wilder, Allan, Power, and Robertson demonstrated that neoplasms of pancreatic islet tissue may produce excessive amounts of insulin and cause hypoglycæmia. The first successful removal of an islet-cell tumour, with complete remission of the symptoms due to hypoglycæmia, was recorded by Howland, Campbell, Maltby, and Robinson in 1929. Since that time the number of successful cases has grown, but islet-cell tumours must still be regarded as rare. Meyer, Amtman, and Perlman (1941) were able to find records of 53 cases successfully treated surgically and we have been able to find records of 9 more cases and add 1 case of our own—Windfeld, 3 cases (1940); Rudd and Walton (1941); Conn and Conn (1941); Quarrier and Bingham (1942); Beek, Haex, and Kooreman (1942); Rayner, Rogerson, and Gaymer Jones (1943); and Lups (1944).

CASE REPORT

HISTORY.—The patient, a schoolboy aged 13 years, was referred to J. MacD. H. by Dr. D. Wallice, of Wrexham, as a probable case of 'petit mal'. During the past twelve months he had had several attacks of disturbed consciousness, occasionally associated with twitching of the limbs and face. The twitching was usually bilateral, but more pronounced on the left side. He did not always lose consciousness during these attacks, but often was unable to speak, and his mother had always noticed a "glazing of the eyes". He was first seen on April 10, 1945, and during the previous month the attacks had become more frequent, occurring about once a week. They had usually been in close relation to meals, either immediately before or after, and occasionally they occurred after exercise. He had never noticed any excessive hunger, but his mother said that his appetite was much greater than usual in a boy of his age. Although the description of the attacks suggested

'petit mal', some of the lapses of consciousness were followed by long periods of deep sleep or even stupor, from which it was difficult to rouse him. This raised the suspicion that hypoglycæmia might be the cause.

The boy is highly intelligent and his own verbatim description of the attacks is interesting: "The first attack which I had was in June, 1944. I was having dinner when my mother noticed that I was dropping the food on the tablecloth and that I was eating hardly any of it. She also noticed that my eyes were glazed. I was taken upstairs and put to bed, where I fell asleep immediately and slept for about three hours. When I awoke I did not remember anything and I only know what happened because I was told. The majority of the fits were not like this one, however. They usually occurred near to a meal time or after I had been running about. During these attacks I could not keep still and I had little control over my limbs. The left side of my body was usually worse than the right. I could usually keep still after a rest of about an hour. After one fit I slept for about five hours and when I woke up I could not remember anything about it. I had one bad attack on the football ground while watching a match. It was a very cold day and I began to feel restless and kept falling back against the people behind me. After watching the match for about half an hour my legs seemed to lose all feeling and I collapsed. I was in a daze and can dimly remember a man moving my arms about to wake me up. I got up and found that I could walk and my uncle started to take me home when I collapsed again, so a taxi was sent for and I was taken home and put to bed, where I slept for two hours. When I woke I could only remember small parts of what had happened to me and thought at first that I had been dreaming. I could not remember seeing any of the football match. I had a headache for about an hour after waking up. When I felt the last fit coming on I had a dose of glucose and I was all right in about fifteen minutes. (This was after the presumptive diagnosis had been made and he had been told to take glucose if he felt an attack beginning.) During at least one of the fits I had difficulty in talking. My teeth were grinding together and my tongue curled up and I could not uncurl it for about five minutes. After lying down for half an hour I recovered."

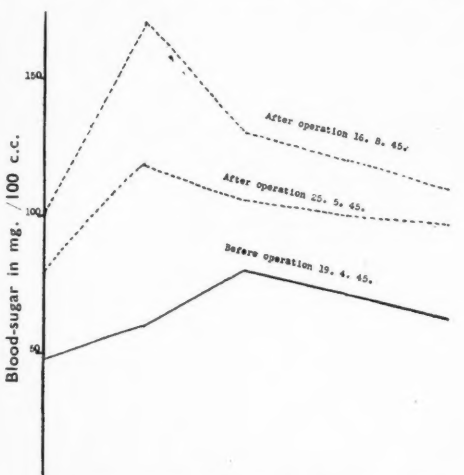

FIG. 365.—Oral glucose tolerance curves before and after operation. 50 g. of glucose given by mouth.

ON EXAMINATION (April 10).—There were no abnormal physical signs of any kind. The B.P. was 125/60. X-ray films of the skull showed no abnormality.

On April 11 the blood-sugar after fasting for twelve hours was 56·5 mg. per cent. He had no fit on the day this estimation was done, but had two more during the week before he was seen again on April 17. An oral glucose-tolerance test on April 19 after a twelve-hour fast gave the following figures:—

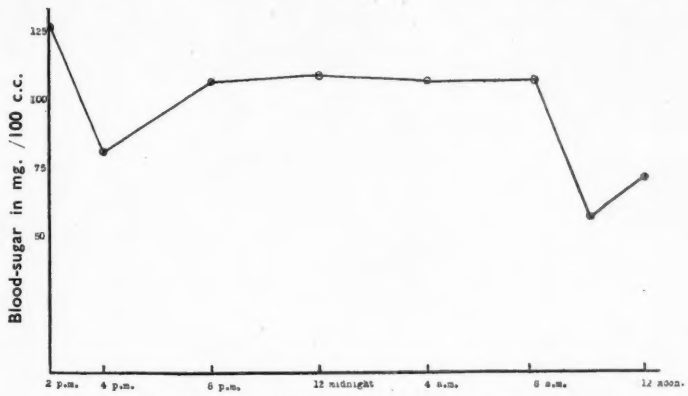

FIG. 366.—Diurnal variation in blood-sugar levels on normal hospital diet.

Fasting blood-sugar	47·5 mg. per cent	
½ hour after 50 g. of glucose	60 mg. „ „	
1 „ „ 50 g. „ „	85 mg. „ „	(*Fig.* 365)
1½ hours „ 50 g. „ „	72 gm. „ „	
2 „ „ 50 g. „ „	65 mg. „ „	

A diagnosis of endogenous hypoglycæmia was thus established and the possibility that it was due to an islet-cell tumour was seriously considered as there was no evidence of any other endocrine disease.

He was transferred to the Staffordshire General Infirmary on May 1 for further investigation, to be followed, if thought necessary, by laparotomy and exploration of the pancreas. No further fits occurred. A fractional test-meal showed no abnormality of gastric secretion and X-ray examination of the alimentary tract showed no abnormality.

On May 2 the diurnal variation of the blood-sugar on the ordinary hospital diet showed low, but not "hypoglycæmic" levels about three hours after the main meals of the day, namely, breakfast and the midday meal (*Fig.* 366).

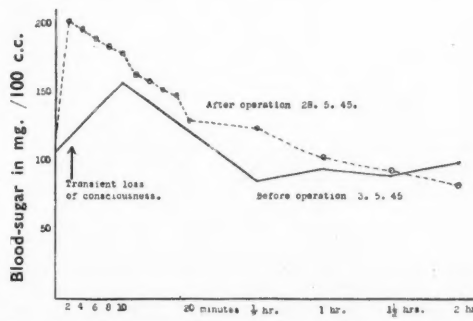

FIG. 367.—Glucose tolerance curves before and after operation following intravenous injection of 10 g. of glucose.

On May 3, on attempting to carry out an intravenous glucose-tolerance test, the boy fainted after 10 g. of glucose had been injected into an antecubital vein. Unfortunately a blood sample was not taken until ten minutes had elapsed, so it is impossible to say whether the transient lapse of consciousness was due to hypoglycæmia, but the test was repeated on May 8 after

injecting 20 g. of glucose intravenously during about three minutes and blood samples were taken at two-minute intervals during the first twenty minutes of the

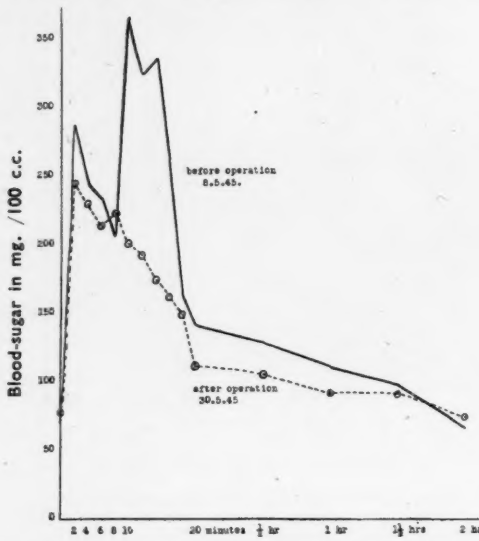

FIG. 368.—Glucose tolerance curves before and after operation following intravenous injection of 20 g. of glucose.

test. An immediate rise in the blood-sugar was observed, followed by a sharp fall during the first eight minutes of the test and then by an even greater rise (*Figs.* 367, 368). These observations were made after a twelve-hour fast. Ten minims of adrenaline injected subcutaneously produced a rise in blood-sugar, reaching a maximum in two minutes, but there was no secondary "kick" in the curve as in the one following the injection of 20 g. of glucose (*Fig.* 369).

Although no conclusive evidence is furnished by these blood-sugar curves, the whole clinical picture strongly suggested a diagnosis of islet-cell tumour and it was decided to perform an exploratory laparotomy. This was done by B. R. S. on May 14.

AT OPERATION.—Under ether anæsthesia the abdomen was opened through an upper right paramedian incision. During the early stages of the operation the pulse became rapid and the breathing irregular, so 40 g. of glucose were injected intravenously. On palpation of the pancreas a tumour about the size of a pea, firmer than the surrounding pancreas, was soon felt in the head. The gland was then exposed through the gastrocolic omentum. There was some difficulty in obtaining a full exposure of the pancreas as the lesser sac did not extend to the right beyond the pyloric antrum. A careful search revealed no other pancreatic tumours. Further examination of the nodule in the head of the pancreas gave the impression that it was partly within the duodenal wall at the level of the ampulla of Vater; so much so that it was decided to attempt its removal through the duodenum, but palpation through an incision into the bowel showed that it was not actually in the duodenal wall. It became obvious that the best approach would be from the posterior surface of the head of the pancreas.

After closure of the incision into it, the duodenum was mobilized and the head of the pancreas exposed from behind. The gland was incised over the tumour, and as the latter did not appear to have a satisfactory capsule it was resected together with some surrounding pancreatic tissue, the rather free bleeding being controlled by fine catgut sutures.

In spite of the risk of damage to the neighbouring ducts a complete removal was thought to be necessary, as the tumour was very small and even a fragment left behind might have been sufficient to cause hypoglycæmia.

A stab drain was left down to the duodenum and the abdominal wound closed.

The boy's convalescence was satisfactory, and as there was nothing more than a little serous discharge from the drain it was removed on the fifth day.

The operation was finished at 12.30 p.m. and at 2.15 p.m. the blood-sugar was 184 mg. per cent, at 3 p.m. 210 mg. per cent, and at 4 p.m. 200 mg. per cent. The following day the blood-sugar levels were as follows: 10 a.m. 141 mg. per cent; 12 noon 109 mg. per cent; 2 p.m. 108 mg. per cent; 4 p.m. 113 mg. per cent. The glucose-tolerance tests, oral and intravenous, which had been carried out in the pre-operative period were repeated under similar conditions during convalescence, and although the fasting blood-sugar still remained at a rather low level it was higher than before. The sharp fall in blood-sugar observed in the 20-g. curve before operation was not found afterwards (*see Fig.* 368).

The boy has now been under observation for five months and no further fits have occurred. An oral glucose-tolerance curve on Aug. 16 gave a curve more nearly normal.

HISTOLOGICAL EXAMINATION OF THE TUMOUR.—The tumour consisted of a roughly spherical mass approximately 1 cm. in diameter, yellowish-grey in colour, with a shaggy surface and hard consistency. It was fixed in formol saline.

The mass may be divided for convenience into an outer and inner zone, but these blend at their junction. The peripheral zone, of variable thickness, consists of acinar cells, occasional islets and ducts, closely resembling, and probably identical with, normal pancreas. This zone extends over approximately three-quarters of the circumference; the remaining quarter shows the inner zone at the surface. The inner zone consists of hyaline connective tissue, in which are set, first, duct-like structures, with a clear lumen, lined by tall columnar epithelial cells, some of which contain material resembling mucin which stains pink with mucicarmine; and secondly, collections of cells closely resembling islet cells, arranged about wide blood-vessels, mostly of capillary type. The outer and inner zones interdigitate where they touch, without clear division. Furthermore, several of the islets

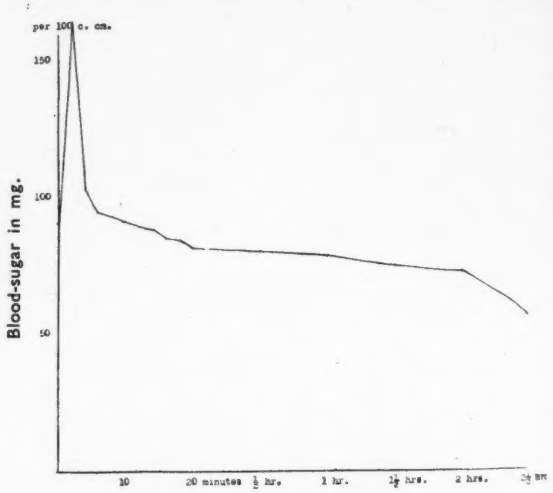

FIG. 369.—Blood-sugar curve following subcutaneous injection of 10 minims of adrenaline.

of the peripheral zone, where they abut upon the inner zone, are unusually large for normal pancreas and form a sort of transition towards the concentrated islet tissue of the centre, where the islet collections and capillaries are much larger still. Again, a few acinar cells are set in the fibrous tissue of the outer part of the inner zone. The ducts of the peripheral zone are set

in trabeculæ of fibrous tissue, as in normal pancreas, whereas the duct-like structures of the centre often immediately abut upon collections of islet cells without interposition of fibrous tissue.

Evidence of malignancy must now be considered. The appearance of islet cells in vessels is probably an artefact, due to section cutting of the spongy tissue of islets. For instance, *Fig.* 373 shows a capillary and islet

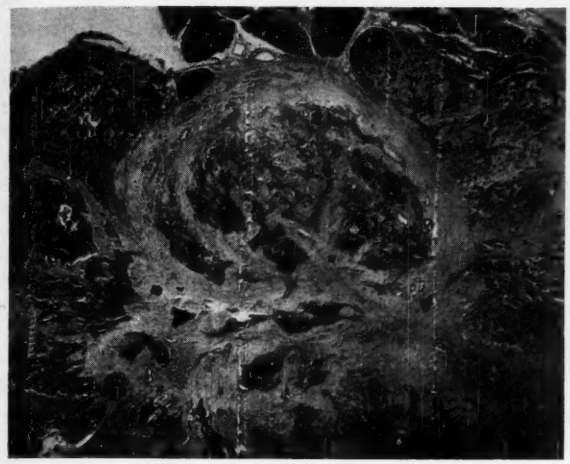

FIG. 370.—General view showing irregular distribution of fibrous tissue. Interdigitation of different cell-groups. Islet-collections seen to touch some large "ducts". (× 12.)

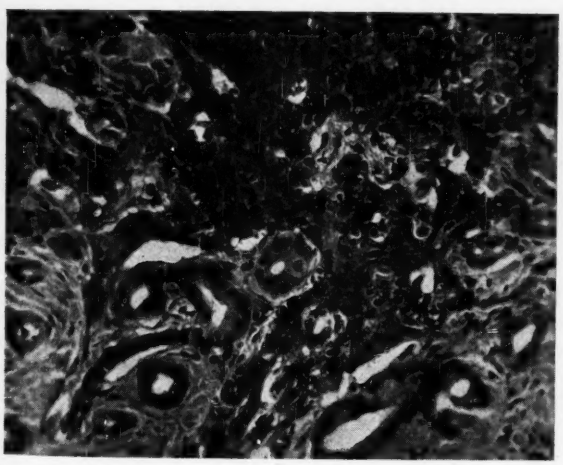

FIG. 372.—Capillaries and islet-collections abutting directly upon some "ducts"; whereas other "ducts" are set in fibrous tissue, as in normal pancreas. (× 165.)

cells inside a duct-like structure lined by columnar epithelium. No mitotic figures were seen. The variations in morphology consist of duct-like structures and islet-like structures, but no anaplastic variations are seen —there is no de-differentiation. On the other hand, the fibrous tissue does not form a complete capsule and the interdigitation of almost or quite normal pancreas and islet masses may be urged as evidence of 'invasion'. Collections of islet cells, unquestionably larger than any seen in normal pancreas, are seen peripheral to the 'capsule'. No compression of surrounding tissue is seen.

DISCUSSION

1. **Diagnosis.**—Although islet-cell tumour is rare, endogenous hypoglycæmia is not uncommon and occurs in a variety of dissimilar diseases. Himsworth (1943) classifies the causes of endogenous hypoglycæmia as follows:—

a. Excessive Removal of Sugar from the Blood.—
- i. Excessive exercise.
- ii. Low renal threshold for glucose.

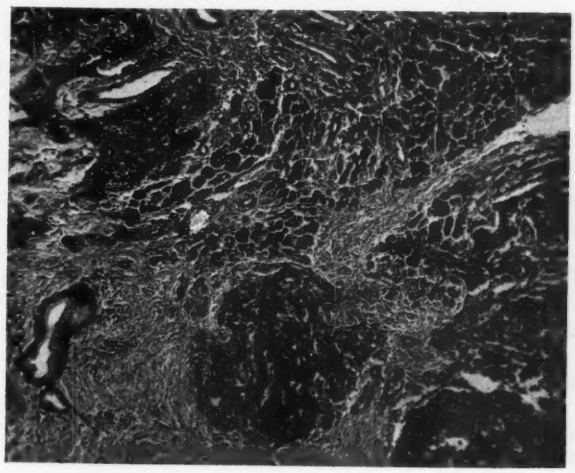

FIG. 371.—Interdigitation of the constituents. "Ducts" —the larger in fibrous tissue, the smaller in islet—collections. Acinar cells are well seen. (× 60.)

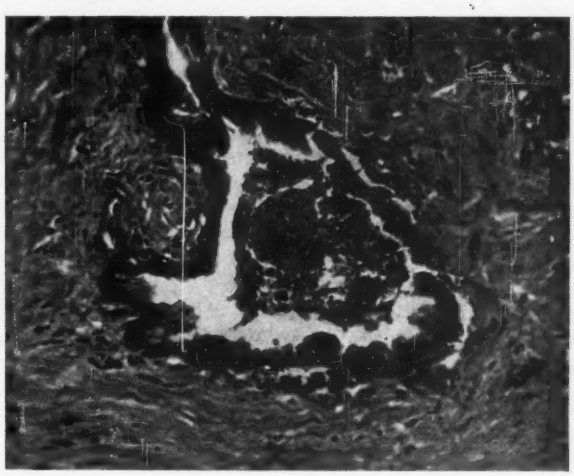

FIG. 373.—"Duct" in dense fibrous tissue. The appearance is thought to explain the "embolism" of various authors. Islet-cells and a capillary in lumen. (× 165).

b. Diseases of the Liver.—
- i. Acute necrosis.
- ii. Certain cases of chronic inflammation.
- iii. Widespread secondary carcinoma.
- iv. Von Gierke's glycogen disease.

c. Disorders of Endocrine Glands.—
- i. Hyperinsulinism: α. Islet-cell tumour; β. Islet-cell hyperplasia.
- ii. Hypopituitarism.
- iii. Addison's disease.

d. Idiopathic Spontaneous Hypoglycæmia.

The clinical diagnosis of endogenous hypoglycæmia should present no difficulty if the clinician is aware of the symptomatology, which is in no way different from that of insulin overdosage. The

symptoms have been thoroughly studied during the past few years since the introduction of insulin therapy into psychiatric practice and may be classified as follows :—

Mild Symptoms.—Fatigue and lassitude, often accompanied by a feeling of faintness and hunger. Epigastric discomfort, sometimes amounting to severe abdominal pain. Sweating, pallor, and vertigo. Drowsiness, tachycardia, palpitation, extrasystoles, and occasionally precordial pain.

Moderately Severe Symptoms.—Any of the above mild symptoms may be present in exaggerated form. In addition, there may be brief mental disturbances or lapses of consciousness resembling ' petit mal '. More florid psychiatric symptoms include depression, euphoria, confusion, and occasionally violent behaviour. States resembling alcoholic intoxication occur frequently. In many cases a disturbance of sleep rhythm occurs and conditions resembling Parkinsonism have been described. Hysterical behaviour, loss of memory, and aphasia have occurred.

Severe Symptoms.—Any of the above symptoms may alternate with attacks of coma, with or without convulsions. Many of the cases described were at first regarded as epileptic. The coma and stupor may be very prolonged and suggest some serious intracranial lesion such as tumour or vascular accident. Violent delirium may occur.

The physical signs are negligible, even during attacks of coma, but occasionally disturbances of tendon reflexes and extensor plantar reflexes are found. It is unusual for any abnormality to be found in the intervals between attacks.

It is usually easy to recognize destructive disease of the liver, Addison's disease, and pituitary cachexia. The greatest difficulty is in differentiating between ' idiopathic ' or so-called ' functional ' hyperinsulinism and the hyperinsulinism due to hyperplasia of the islets of Langerhans or islet-cell tumour. Direct evidence of the existence of islet-cell hyperplasia is difficult to establish. There are many records of hyperinsulinism having been relieved by partial pancreatectomy, although no islet tumour was found. Some of these case have been regarded, perhaps on slender grounds, as examples of islet-cell hyperplasia. The small size of islet-cell tumours and their not infrequent multiplicity make discovery and surgical cure difficult. It should be borne in mind that they may lie outside the pancreas, as in the case reported by Rudd and Walton (1941). If our submission that these tumours are in the nature of heterotopia be correct, search for them should include a much wider area than the pancreas and embrace the duodenum, jejunum, and stomach, where pancreatic heterotopia has been shown to occur.

From the point of view of clinical behaviour J. W. Conn has divided endogenous hypoglycæmia into two broad groups—stimulative hypoglycæmia and fasting hypoglycæmia. In ' functional ' hyperinsulinism the stimulative type of hypoglycæmia is said to occur. A precipitous fall in blood-sugar follows the ingestion of large amounts of carbohydrate. The level of the fasting blood-sugar, however, remains normal, even when the dietary carbohydrate is restricted. The transient postprandial attacks of hypoglycæmia are controlled by reducing the carbohydrate content of the diet. The fasting type of hypoglycæmia is well illustrated by certain types of liver disease in which restriction of the dietary carbohydrate produces a low level of the fasting blood-sugar. A diet rich in carbohydrate controls this type of hypoglycæmia.

In organic hyperinsulinism, according to Conn and Conn (1941), both these types of hypoglycæmia occur. A low level of the fasting blood-sugar is always found and restriction of dietary carbohydrate depresses it further. At the same time, however, a stimulative effect may be seen in the fact that two to four hours after a meal the level of the blood-sugar is frequently far below that observed in the post-absorptive state. These authors regard their observations as evidence of an excessive stimulation, by the ingestion of carbohydrate, of the mechanisms involved in the rapid removal of sugar from the blood. They found in their case of islet-cell tumour, that the fasting hypoglycæmia, obviously the result of too much insulin, was associated with abnormal inhibition of glycogenolysis in the liver and with excessive combustion of sugar. The stimulative effect disappeared after removal of the tumour.

In our case there was a fasting hypoglycæmia and the diagnosis, at first only suspected, was more strongly suggested by this finding. There was also a ' stimulative ' effect about three hours after meals, as shown by the variations in the blood-sugar throughout the day when the patient was taking a normal hospital diet (*see Fig.* 366). It is also tempting to assume that the transient loss of consciousness following the intravenous injection of 10 g. of glucose was the effect of ' stimulative ' hypoglycæmia, particularly in view of the fall in blood-sugar observed in the first few minutes during the later test using 20 g. of glucose. Definite conclusions from these isolated observations on a single case are impossible and repetition in further cases of islet-cell tumour is obviously necessary before their value can be assessed. We have been unable to observe the initial fall in blood-sugar in the 20-g. curve in six epileptic patients who had no fasting hypoglycæmia. The oral glucose-tolerance curves before and after operation are of the flat type often seen in mild diabetes, although they are at a lower level. After a time the flattening disappears. This flat type of curve is not uncommon in hyperinsulinism, but in the present state of our knowledge it cannot be said that there is any infallible diagnostic procedure in cases of islet-cell tumour.

2. Pathology.—It is submitted that the explanation in this case, and in many other published cases, lies in heterotopia—*anlage*—with possibly some degree of benign neoplastic proliferation. Nicholson (1921 onwards), as Professor Stewart observed, " almost single handed " in this country, studied the relationship between heterotopia and new growth (Stewart and Taylor, 1925). As we understand it, Nicholson maintained that there is " no borderland between malformations and tumours " and again " typical tumours come very close to malformations "; " . . . the displaced cells undergo their normal differentiation . . ." It is noted that accessory pancreas is found between the layers of mesentery and within the wall of stomach or duodenum. A carcinoma of such accessory pancreas is described—hamartoblastoma, but " the truth

appears to be that cell-nests usually undergo differentiation together with the other tissues of the body. In rare cases they fail to do so. Tissue malformation or differentiated cell-nests are not predisposed to tumour formation ".

Stewart and Taylor (1925) state that heterotopia of pancreas occurs in the subserous or submucous coats of the duodenum (also stomach and jejunum); islets are seen in some cases (Stewart and Hartfall, 1928). They describe an adenomyoma of the stomach containing pancreatic type of epithelium; undifferentiated duct-like structures are also figured. The blending of the various glandular groups of normal gut wall and pancreatic heterotopia resembles the findings in our case. Plain muscle was prominent—almost absent in our material. It is stated " . . . specimens of differentiated heterotopic pancreas not infrequently show small areas of undifferentiated duct-like tissue obviously in excess of duct requirements. . . ." The important statement is made that " malignant tumours of accessory organs are extremely rare ". Now this seems vital to the discussion on the evidence of malignancy in " adenoma " of the islet cells.

It appears to be established that islets arise in the embryo directly from duct-like (undifferentiated) structures. Barnard (1932) suggests that " the adenoma has replaced some of the pancreatic tissue proper and that fibrous tissue and ducts have persisted ". Whipple and Frantz (1935) describe " . . . duct-like structures. Whether they are to be interpreted as adult ducts from the original pancreatic tissue or new-formed tumour ducts it is difficult to say. The point is rather an academic one, since the origin of islet cells from duct epithelium has been demonstrated "—the suggestion is here made that further discussion is academic. It is submitted that this is not so and that the views herein set forth are very material to the issue, especially with regard to malignancy.

To summarize: (1) The mass does not consist merely of islet cells, but includes " ducts " and cells resembling the external-secretory cells of the pancreas. (2) The position, in contact with the duodenum, is consistent with the embryological origin as a duodenal bud. (3) In the normal pancreas ducts are surrounded by bulkheads of fibrous tissue. Here islet collections often abut upon " ducts " without interposition of fibrous tissue. It seems unlikely that such fibrous tissue should disappear, and if so, suggestions that the duct-like structures are remains of pancreas invaded by tumour seem ill-founded. Again, the duct-like structures are too numerous to satisfy the conditions contemplated by such a view. The absence, often remarked by authors, of complete encapsulation in these tumours, and the interdigitation of almost normal pancreas with ' adenoma '—often deemed evidence of invasion—are unlike the findings in adenomata elsewhere; at the same time the absence of signs of de-differentiation or anaplasia, and the usually excellent results (except in cases showing metastases) of operative removal, in spite of ' tumour cells ' seen in vessels, are hardly consistent with carcinoma.

Conclusion.—It is submitted that cogent evidence has been adduced to show that ' adenomata ' of the islets of Langerhans are of the nature of heterotopia and that this view explains the presence of ' ducts ' and ' invasive ' growth and the ' absence of encapsulation ' which have perturbed histologists inclined to regard the masses as true adenomata of islet cells.

SUMMARY

A case of hyperinsulinism due to a pancreatic islet-cell tumour is described. The cerebral symptoms of the patient were entirely relieved by removal of the tumour. Certain features of the response to intravenous injection of glucose are described which have not hitherto been noted.

Histological evidence that these tumours are in the nature of heterotopia is presented.

Our grateful thanks are due to Professor M. J. Stewart and Professor H. P. Himsworth for helpful advice and criticism, and to Mr. T. C. Dodds for the photomicrographs.

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THE PRESENT POSITION OF SURGERY IN THE TREATMENT OF BLEEDING PEPTIC ULCER*

By GORDON GORDON-TAYLOR, C.B., O.B.E., Surgeon Rear-Admiral

Some apology is demanded from one who would venture yet again to write on this controversial subject, but the problem has interested me for a quarter of a century, and my own attitude towards the question has altered more than once during the years that have elapsed since the last Armageddon. I offer no excuse for changes in outlook that have from time to time controlled my scalpel, for no surgeon worthy of the name remains intransigently and adamantly bound to some particular line of therapeutic conduct which he may have cherished or championed heretofore, nor should his attitude towards any problem be crystallized beyond the power of reasoned persuasion; greater knowledge, and widened experience laudably modify opinions that formerly held sway.

The problem of the surgery of hæmorrhage from chronic peptic ulcer was discussed at considerable length by the writer (1937) in a previous number of the British Journal of Surgery, to which the reader is referred. To many of the opinions expressed therein the author still adheres; a natural repugnance to mere repetition and the restrictions of space imposed by paper shortage suggest reference to previous communications by the writer (1935, 1937, 1939) on the subject, but there are other former views which require modification in the light of the more recent results of competent modern medical therapy.

Even before the outbreak of the present war, surgical interest in ulcer-hæmorrhage had fortunately produced certain important repercussions, including a re-awakening of medical enthusiasm over the treatment of this group of patients, greater vigilance and alertness in the medical observation and conduct of such cases, the exploration of new methods of assessment of the amount of bleeding in progress and its effect on the organism, the scientific employment of replacement-fluids of various kinds, including blood, and an earlier recourse to gastric feeding.

Therapeutic opinion still varies as to the amount of blood which should be administered by drip transfusion; computations of blood volume are now appraised as of greater accuracy than hæmoglobin readings in estimating severity of blood-loss in the early hours of the cataclysmic catastrophe. Especially in recurrent hæmorrhage, an estimate of the calibre of the artery bleeding in the base of the ulcer has sometimes been tentatively adumbrated by means of repeated blood-volume analysis, and more than an occasional appeal to surgery from the physician has been thereby evoked. The efforts of medical colleagues, more especially those under whose care in the Courtauld Research Wards of Middlesex Hospital during normal peace-time conditions were segregated many of the severe ulcer hæmorrhages, have nowadays reduced "medical mortality" to a far smaller percentage than heretofore, so that the greatest judgement and caution must necessarily be exercised before pressing surgical intervention for patients overtaken by this alarming complication of a gastric or duodenal ulcer. Surgery required during active ulcer hæmorrhage indubitably carries least risk in the early hours of the bleeding; nevertheless, although Finsterer's "third day" may possibly be a critical period for operation, the successful results obtained by several of my surgical friends from interventions carried out at a later date in patients suffering from continued or recurrent bleeding belie the universality of the Austrian surgeon's dictum. The evidence seems to attest speed as an important factor in determining the issue of any surgical adventure for the control of gastric hæmorrhage; the extended experience of operators on both sides of the Atlantic fails to confirm the absolute inseparability of success and the employment of regional anæsthesia alone, as emphasized by Finsterer, Ferguson, myself, and others.

The purpose of this lecture is not to promulgate my own personal experience or to secure converts to any surgical *penchant* of mine in past days regarding these cases, but rather to paint a picture illustrating the results of the best modern therapy gleaned from the recent experience of many physicians and surgeons of many institutions. To those who have within the past few months so very kindly permitted me access to their notes and figures, who have given their time to discuss the problem with me, who have often with great trouble to themselves collected and collated information on the group of cases on which I desired their aid, my gratitude is deep and heartfelt.

The recent material which has been made available to me has emanated from certain of the voluntary hospitals of London, from hospitals of the London County Council and of the Middlesex County Council, as well as the larger Royal Naval Hospitals in this country; upwards of 1700 cases of ulcer hæmorrhage have passed under review for this address.

I have endeavoured for the lecture to survey the problem from a new angle, studying carefully the more recent statistics of physicians treating cases by non-operative measures, scrutinizing the histories of fatal cases where these have been available, and conjecturing what percentage, if any, of those who died might possibly have been saved had surgical measures been resorted to when non-operative treatment seemed and, in fact, proved unavailing. It becomes at once apparent how greatly the results of the non-operative treatment of gastroduodenal hæmorrhage of ulcer origin have improved in the past ten years. Dr. Izod Bennett and his medical 'team' at Middlesex Hospital

* Being the First Lettsomian Lecture, Medical Society of London, July 18, 1945.

carefully classified (1938) cases of ulcer hæmorrhage admitted to the Courtauld Research Wards by means of blood-volume estimations. Their "*severe cases*" were those who had lost over 50 per cent of their total blood-cells; in 1935 the mortality in this group was 27 per cent, and in 1936 43 per cent; whereas in 1937 only 5 per cent died, and in 1938 only 11 per cent. In Bennett's own words, "*blood transfusion, correctly applied, is the most important single factor in saving life in gastroduodenal hæmorrhage.*" Diligent scrutiny of the data concerning the 10 cases of this series who died as a result of ulcer hæmorrhage shows that 9 of them were suffering from chronic ulcer, and 1 from acute ulceration. One of the cases was under 50 years of age, and none under 40.

Of the 10 deaths of the series it is conceded by the physicians that 5 rapidly succumbed after admission to hospital purely from loss of blood, and, in the opinion of those in charge, had it been possible in the time available to have replaced the blood lost, some, if not all, of the fatal cases might have survived. Three other patients of the same group died from bronchopneumonia—so often, of course, a terminal event in the later stages of protracted bleeding, which might dubiously have been arrested by timely operation. Yet it is my considered opinion that surgery, if attempted, could only have saved at most one or two of the cases in Bennett's series that succumbed.

I am under a very special debt of obligation to Dr. Avery Jones, of the Central Middlesex County Hospital, who furnished me with an analysis of 389 consecutive admissions for gastroduodenal hæmorrhage from June, 1940, to January, 1945, of whom 34 died, i.e., 8·7 per cent mortality. There were 87 ulcer hæmorrhages from chronic gastric ulcer with 12 deaths, i.e., 14 per cent mortality. There were 110 ulcer hæmorrhages from duodenal ulcer with 9 deaths, i.e., 8·1 per cent mortality.

But Dr. Avery Jones afforded me still further assistance and most courteously provided me with notes of the fatal cases of ulcer hæmorrhage from June, 1940, to January, 1945, inviting my criticism. These fatal cases he had classified into: (*a*) Cases in which surgery or further surgery seemed impracticable. (*b*) Fatal cases despite an operation to control hæmorrhage. (*c*) Fatal cases which in his opinion might have profited by surgery.

a. Of 16 deaths which belong to Avery Jones's first group, a surgeon's critical attitude fails to convince me that operation could, or would, have saved any of that group. It is just within the bounds of possibility that if an immediate gastroduodenal resection had been performed for 2 cases which suffered from concomitant perforation and hæmorrhage instead of a mere suture of the perforation, these might have been saved. Another woman of 57 in the series might also have been a 'possibility', but the scales were certainly heavily loaded against a man of 67 who had been admitted for a fracture of the neck of the femur three weeks before his duodenal ulcer bled and perforated (!) I find little to support the view that any surgery would have bettered the results in this sub-group of fatal cases.

b. Cases operated on for hæmorrhage who died—all these were of a 'forlorn' type, and their deaths call for no criticism or adverse comment from a surgeon.

c. Cases which, according to Dr. Avery Jones, might have profited by surgery. Of 7 in this category, possibly 1 case, a male of 56, might have been saved by surgical treatment. Various considerations relating to the other cases in this group convince me that surgery was unlikely to save any. Dr. Avery Jones has wisely expressed his special anxiety over cases of chronic gastric ulcer who undergo recurrent hæmorrhage while in hospital and who are over 50 years of age. In his series of cases 10 fell into this category and 6 died (60 per cent). This able and experienced physician is led to the conclusion that more help should be obtained from the surgeons for this group of patient. In his opinion, clinical observations of grave significance in connexion with chronic gastric ulcer are:—

1. A thickened tortuous radial artery.
2. Age over 50—5 deaths out of 18 (28 per cent).
3. Severe pain preceding hæmorrhage—5 deaths out of 12 (40 per cent).
4. Pain persisting after hæmorrhage—2 deaths out of 4 (50 per cent).
5. Recurrent bleeding: Once—6 deaths out of 12 (50 per cent); Twice—5 deaths out of 8 (60 per cent). He writes: "By selecting cases which come partly or wholly within this group a reduction in mortality from bleeding chronic gastric ulcer might be effected by early operation".

In 1936 I was involved in a debate on the treatment of hæmatemesis at the Medical Society of London. Sir Henry Tidy (1937), with whom I crossed swords on that occasion, gave an account of 30 cases of 'grave' or 'moderate' hæmatemesis or melæna admitted to his wards at St. Thomas's Hospital during the years 1931–1936 inclusive. Of Tidy's cases 5 died, but 3 of these had accessory pathological conditions which indubitably rendered them little likely to respond favourably to either medical or surgical treatment. Of the remaining 27 cases, the help of the surgeons was invited in 2 cases, in 1 of which an unsuccessful operation was undertaken; in the other the surgeon declined to intervene. A remaining case of this series was operated on by another surgical team, a jejunal ulcer was dealt with and bled again, though the patient recovered; this was a case which would almost certainly have got well without surgery. In this series Tidy definitely proved his point that further surgery in his particular series of patients would *not* have improved the recovery-rate.

Thanks to the kind assistance of D. A. J. Ebrill, I was provided with information concerning 81 cases of ulcer hæmorrhage admitted to St. George's Hospital from 1940 to 1944, both years inclusive. The mortality-rate was 11 per cent, and of the 9 cases who died all were patients in the "late sixties and the seventies", except 2 cases. One of the latter was a man of 42 who had a perforation and soon after suffered a hæmorrhage, which proved disastrous: the fatality of this combination, as will be emphasized in a subsequent paragraph, is proverbial. The other was a man of 45 years who

survived four days in hospital before succumbing from hæmorrhage; possibly in his case surgery might have helped, but no post-mortem was possible to afford further information. It is very dubious, therefore, if the employment of surgery would have made any difference to the results in this series; the age of almost all the fatal cases would appear to weigh heavily against any prospect of surgical success.

Thanks to the kindness of Dr. E. D. H. Cowen, I have been vouchsafed information about 15 cases of gastroduodenal hæmorrhage admitted to Westminster Hospital in eighteen months. A critical study of the fatal cases persuades me that in only 1 case might surgery have altered the result, and even that patient might well have been termed a 'very bad surgical risk'—a male, aged 61, was found unconscious in a pool of blood, but survived in hospital for four days, during which he had further attacks of hæmatemesis and required suprapubic drainage for retention of urine. At the autopsy there was found a gastric ulcer at the pyloric end of the stomach, measuring 5 in. × 1¾ in., which had eroded the splenic artery.

The effect of age on the mortality rate of ulcer hæmorrhage has already been mentioned, and will be referred to in a subsequent section. This is well illustrated in the figures from the Hillingdon County Hospital very generously placed at my disposal by Dr. Cyril Barnes, of the Medical Unit, St. Mary's Hospital. Under 50 years of age, the mortality in his series was only 3·5 per cent; over 50, 13·4 per cent. Under 60 years, the mortality was 5·7 per cent; over 60, 16·6 per cent. There were 23 deaths in the total series, of which 5 were undoubtedly moribund on admission and 3 other fatal cases were for various reasons extremely 'bad surgical risks'. Nevertheless, 18 cases in the series died from blood-loss despite transfusion; it is true that some of these fatal cases due to blood-loss had been seen by surgeons, who declined operative aid. The percentage of all the ulcer hæmorrhages admitted to the Hillingdon Hospital that were submitted to surgery was strikingly small—only 4 cases out of 275—yet the operators may be said to have 'done their stuff', since 3 of their patients recovered. Still, a surgical critic is left with the uneasy feeling that possibly, even probably, in this series, a more extensive employment of surgery might have saved a few of the 17 that died of blood-loss, and the total recovery-rate might have been raised to an even higher percentage than the creditable figure recorded.

Mr. Norman C. Tanner, of St. James's Hospital, Balham, has generously placed at my disposal an analysis of 360 cases of ulcer hæmorrhage admitted to his wards between May, 1941, and March, 1945. There were in all 40 deaths, i.e., the total mortality-rate of ulcer hæmorrhage was 11 per cent. Tanner divides his series into three periods:—

a. A period when urgent operation was avoided whenever possible, and if performed was almost always a late intervention for persistent bleeding. The percentage of cases operated on during this first period amounted to only 5·2 per cent, and surgery was resorted to on an average sixteen days after admission. The operations resulted splendidly in 9 out of 10 recovering, and included 7 gastrectomies, 1 ligature of blood-vessels supplying the ulcer, 1 closure of perforation in patient bleeding, all recovering. The 1 operative fatality followed a gastro-jejunostomy with tampon of a bleeding duodenal ulce . The total mortality in the group, whether medically or surgically treated, was 9·8 per cent, and the average age of the fatal cases was 62 years.

b. A period *where operation was more readily undertaken*; the percentage of cases operated on was 15—not a very 'staggering' percentage. In this series, operation was undertaken much earlier—usually 36 hours after admission: all the operations were gastrectomies, but 4 died out of 9. The mortality for the whole group, whether treated by non-operative measures or by surgery, was 20 per cent, doubtless so high because the number of aged patients in the group was much greater (33 per cent were over 60).

c. A period where *operation was performed relatively early*, but *where the simplest surgical methods for arresting hæmorrhage were employed.* The percentage of cases operated on was about 10·5 per cent, and the average time of operation was 3·7 days after admission. Of the patients submitted to surgery, 8 lived out of 11 (73 per cent recovery rate). A very remarkable case in this group was an "atheromatous" male of 79 years *in extremis* from his third collapse from duodenal hæmorrhage, whose blood-urea was 140 mg., and who was operated on on the fifth day. The mortality of this third group, whether medically or surgically treated, was only 8·5 per cent. The average age of the fatal cases was no less than 66 years.

It will be seen from these analyses from different institutions that wise and skilled medical treatment and good surgical judgement have paid fine dividends. H. C. W. Nuttall, of Liverpool, in 11 years found it necessary to operate upon 16 cases of profuse ulcer hæmorrhage, of whom all recovered except one patient who had also a perforation of the bleeding duodenal ulcer. My friend H. W. S. Wright has saved 3 out of 4 'late' recurrent ulcer-hæmorrhages of desperate character by urgent surgery, while bleeding was still in progress. At one large Naval Hospital from 1939–1944 inclusive there were 2 fatal ulcer hæmorrhages out of 69 cases of varying severity, some very slight. One death was a case of jejunal ulcer, the other a large duodenal ulcer in a man of 46, who continued to bleed, received 12 pints of blood during his sojourn in hospital, and died on the tenth day. In neither case was a surgical opinion invited until the day before death, when operation would merely have been wasteful of time. At another large Naval Hospital there was only 1 death among 45 ulcer hæmorrhagés, yet that fatal case was in hospital for three months with recurrent hæmorrhage, and received in all 20 pints of blood, but no surgical aid was vouchsafed him (!) On the other hand 2 recurrent ulcer hæmorrhages were saved by urgent operation at Haslar, and another case of recurrent hæmorrhage from a chronic ulcer on the lesser curve of the stomach, under treatment in an Auxiliary Naval Hospital, was

brought to a successful issue by the surgical enterprise of Surgeon Lieutenant-Commander E. H. Murchison, R.N.

Ivor Lewis, from his extensive experience at the North Middlesex County Hospital, Edmonton, is inclined to think that the desirability of surgical treatment should at least be discussed in all patients in whom the hæmoglobin is under 40 per cent and who are over 40 years of age. Recently, out of 7 consecutive surgical interventions by this able surgeon for ulcer hæmorrhage all recovered; the patients were all over 40 and had suffered a second or third recurrence of bleeding during the 'hæmorrhagic phase'. In 5 a partial gastrectomy was performed; in 2 an elliptical resection of an ulcer of the lesser curve.

Perhaps the most wonderful series indicating the value of co-operation between physician and surgeon in the treatment of ulcer hæmorrhage is that kindly furnished me by Chase Farm E.M.S. Hospital. The figures are concerned with the results of treatment of 81 cases presenting evidence of considerable gastroduodenal bleeding admitted to hospital between 1940 and February, 1945; in 50 of these the hæmoglobin had fallen below 50 per cent, and in 4 below 25 per cent. Four cases in the series died, i.e., a mortality-rate of less than 5 per cent, but this fatality-rate would doubtless have been much higher—possibly doubled, perhaps trebled—had it not been for the excellent judgement of Dr. Allan Birch, who transferred a number of cases to his brilliant surgical colleague, R. L. Galloway. Of 11 surgical interventions 11 lived. This series shows *par excellence* the achievement of a physician and surgeon working in close unison.

A PERSONAL SERIES OF OPERATIONS DURING THE PERIOD OF HÆMORRHAGE OF CHRONIC ULCER

In the preceding pages I have largely quoted the experience of others chiefly obtained during the recent war years, but I am also anxious to place before readers a personal experience of almost a surgical lifetime comprising 71 operations performed *à chaud* during the hæmorrhage period for bleeding chronic peptic ulcer. The appended table brings the list of my interventions up to date; the cases seem divisible into four separate periods of surgical activity characterized by varying zeal on my part in the employment of operative methods in the treatment of ulcer hæmorrhage. The table does not include interventions for the hæmorrhage of acute ulcer, since surgical undertakings for the bleeding of an acute peptic ulcer date back to a distant past, and the writer has nothing to add to information contained in previous communications. Drip transfusion nowadays renders operation almost invariably unnecessary in this type of case; nevertheless, even to-day rare fatalities are known to occur from the bleeding of acute peptic ulcer.

In the period 1933–39 I may have been swayed a little unduly towards surgery by reason of the dismal results of the non-operative treatment of ulcer hæmorrhage during the preceding period 1926–33, as evidenced by my own personal experience and results emanating from the non-operative measures for ulcer hæmorrhage among in-patients in the Middlesex Hospital and certain other institutions about that time, when the death-rate from hæmorrhage from proved ulcer cases treated by non-operative measures without or with only scanty and sporadic transfusion amounted to upwards of 24 per cent.

Table I.—Summary of 71 Operations performed during Hæmorrhagic Period for Bleeding Chronic Peptic Ulcer (Personal Series)

Period	Operations	Recoveries	Deaths	Percentage Mortality
A. 1919–24	22	20	2	9
B. 1924–26	10	6	4	40
C. 1926–33	10	8	2	20
D. 1933–39	Early operation 18	17	1	5·5
	Late operation 11	7*	4	36
	Total 29	24	5	17
	but amended 27 (2 hopeless cases excluded)	24	3	11

* Two of the four fatalities were hopeless cases.

During this period 1933 to 1939 I was again vigorously exploring surgical methods in an endeavour to improve the results in ulcer hæmorrhage, and our therapeutic armamentarium was reinforced during that period by the introduction of the "continuous intravenous blood-drip" by Marriott and Kekwick. At this time I dealt surgically with 29 patients, of whom 18 were early cases, many being operated upon within twenty-four hours of the onset of bleeding. Only 1 of these succumbed; death in that case was said to have been due to bronchopneumonia, probably related to the presence of some very septic teeth, perhaps to the general anæsthetic and the too liberal administration of morphine. The mortality in this series of early operations was therefore 5·5 per cent.

Of the 'late cases' operated upon in the period of six years preceding the outbreak of the present war no less than 4 patients died from various causes; some of these were 'forlorn hopes', and late operation in my experience has been fraught with a far heavier mortality than early surgery in this class of case.

THE PRELIMINARY MANAGEMENT IN CASES OF HÆMATEMESIS AND MELÆNA

This has been discussed by me in previous communications; it suffices merely to reiterate three recommendations already made:—

1. In every case of hæmatemesis or melæna, whatever the cause, immediate steps should be taken to make contact with a transfusion organization.

2. It is desirable that every case of hæmatemesis of any severity should be transferred to a fully organized and equipped institution. An exsanguinated patient can be moved with the drip transfusion running, when this seems indicated (' *travelling transfusion* ').

3. Early consultation between physician and surgeon, for which this lecture is a plea.

INDICATIONS FOR URGENT SURGERY DURING THE HÆMORRHAGE PHASE

It cannot be insisted too strongly that the diagnosis of a chronic peptic ulcer must be established beyond a doubt before any surgical treatment for ulcer hæmorrhage is even thought of, since a motley collection of pathological conditions may share hæmatemesis as their prominent symptom. Urgent surgery, when employed to control the bleeding of a peptic ulcer, must only be directed against that alarming complication of an ulcer, the diagnosis of which is definitely established. The circumstances of the operation and the limitations imposed by the employment of local anæsthesia, should the latter be selected, preclude energetic abdominal exploration, and if surgery be deemed necessary for a bleeding peptic ulcer, the surgeon must be sure, be quick, and be gentle.

1. Immediate operation is indicated and may on rare occasions be practicable in cases of terrific bleeding, when it appears obvious that some large vessel has been eroded.

2. Urgent operation is required in the rare cases of concomitant hæmorrhage and operation (*see below*).

3. The possibility of urgent surgery should always be considered in patients over 50, especially males with a known chronic peptic ulcer, and perhaps more particularly a chronic gastric ulcer. In this group surgery is still more deserving of consideration, if the patient has a thickened, tortuous radial artery, if severe ulcer pain preceded the hæmorrhage, if pain persists after the hæmorrhage, or if recurrent hæmorrhage takes place in a patient under appropriate medical control.

4. The anxieties engendered by recurrent hæmorrhage in the above group suggest an appeal to surgery, since in Avery Jones's cases of chronic gastric ulcer in men over 50, half the patients died after one recurrent hæmorrhage and 60 per cent after a second bleeding.

5. Where pyloric or duodenal stenosis is present, or mid-gastric narrowing is considerable, hæmorrhage is more likely to persist or quickly recur, and the pathological condition may determine a decision to intervene surgically.

DECISION TO OPERATE

Lest the reader should entertain the opinion that I am recklessly prejudiced in favour of surgery, I would venture to repeat what was written by me eight years ago: " Every case must be considered on its own merits; there can be no hard-and-fast rules. Antecedent disease and intercurrent illness, the previous habits of the patient, his physical conformation, and all that constitutes a sufferer a ' good or bad surgical risk ' must be weighed carefully; the personal courage of the patient as well as of the operator, the environment of the case, the experience and technical skill of the surgeon, are all factors which not only should influence the decision as to the desirability of surgery, but may also indicate the appropriate technique when the abdomen has been opened." R. L. Galloway, of Chase Farm Hospital, believes that operation should only be carried out by someone " who is nimble with his hands and can carry out the operation at the maximum possible speed ". Ivor Lewis, of North Middlesex Hospital, thinks that no surgeon should operate on a case of bleeding ulcer during the ' hæmorrhagic phase ', whose mortality for ' cold ' gastric surgery is more than 5 per cent. This opinion coincides exactly with that of his colleague, W. J. Ferguson, of the West Middlesex Hospital, one of the finest gastric surgeons whom I have been privileged to watch.

THE NATURE OF THE SURGERY EMPLOYED

Although those surgeons who have had experience of operation during the period of acute hæmorrhage from chronic peptic ulcer exhibit a proclivity to the performance of partial gastrectomy, I am most anxious to protest that the treatment of ulcer hæmorrhage in my own hands is not synonymous with gastrectomy. No one technique is appropriate to every case for which operation has been judged necessary. The various surgical measures which I have myself employed have been described and illustrated again and again in previous papers, and I have nothing to add except to describe and illustrate Judin's technique of dealing with a bleeding penetrating duodenal ulcer.

The ' snail-like ' method of closure of the duodenal stump (technique *en escargot*) is strongly recommended by him in cases of acute hæmorrhage from large penetrating callous ulcers of the duodenum where a complete defect of the posterior duodenal wall exists. A free mobilization of the anterolateral wall of the duodenum is imperative, including the division and separation of all adhesions and adventitious membranes immuring the gut at the back of the abdomen beneath the gall-bladder or hepatic flexure. The anterolateral wall must be freed distal to the ulcer or stenosis for at least 4 to 6 cm., not only along the whole upper horizontal first portion of the duodenum, but also along the ' angle '. The precise technique of inserting the sutures can be seen in the accompanying illustrations (*Figs.* 374, 375) which are from Judin's own pen. The particular merit of the method lies in the use of the bunched-up duodenal stump to tampon a gigantic ulcer penetrating into the head of the pancreas. Judin's only apprehension, when first he devised this technique, was lest the inner catgut suture fixing the two or three twists of the ' snail ' should undergo premature digestion, permitting the ' snail ' to uncurl and escape through the superjacent layer of sutures. The Moscow surgeon's experience of the method now amounts to upwards of a hundred cases over a period of ten years, and only once in the earliest year of its essay did this untoward accident occur.

My own experience of this method of closing the duodenal stump is necessarily small, but I have

found it valuable in difficult cases of penetrating duodenal ulcer; however, the opportunity of employing the technique in the treatment of acute hæmorrhage from a duodenal ulcer has so far been denied me.

Sir Henry Tidy (1944) has pointed out the 'territorial' differences in the character and *locale* of peptic ulcer, not only between this country and the Continent, but also between London and England and Scotland; he has also stressed the importance of social environment in this connexion, and has uttered a timely warning about the criteria of satisfactory medical treatment and a reminder that when a first course of medical treatment fails, a second course is often successful. 'Territorial' considerations may therefore play some role in explaining the numbers of operation for acute hæmorrhage performed by Finsterer, Judin, and others. Judin (1943), however, has gloomily described his feelings as a surgeon of a famous special Emergency Hospital, when for the thirtieth, fortieth, and even the fiftieth time it has been his misfortune in the post-mortem room to view the erosion of an artery in the crater of a large ulcer, yet quite accessible for a surgical operation. It is not difficult to understand his surgical bias in view of this experience.

The famous Moscow surgeon has performed 134 gastroduodenal resections *during acute hæmorrhage*, avoiding operations upon very old persons, "except those who looked younger than they really were". There were 21 deaths in this series, but 4 'casual' fatal issues might be excluded from the computation; thus, a woman died six days after operation of infarction of the myocardium; a man died a week after gastrectomy with sepsis and profound jaundice, autopsy revealing purulent calculous cholecystitis, an obstruction of the common duct by a stone, and gas infection of the liver and spleen. Two other patients died owing to faults in technique connected with blood transfusion: one from air embolus, noted during the transfusion and confirmed at the necropsy; the other perished from typical hæmolysis.

Judin considers separately a group of "desperate operations", when after frustrated hopes for recovery by means of conservative treatment or "hæmostatic transfusions" the patients were operated on, always without pulse, almost always unconscious, and in such degree of anæmia, that expectations of recovery were founded, not so much on operation, as on some miracle. He gives a few detailed examples of these cases, in which there were 9 deaths in 23 gastrectomies. Without these "desperate operations" the great majority of such patients would almost certainly have perished.

The 'forlorn hope' ulcer hæmorrhage, for which appeal, perhaps belated appeal, is made to surgery is sufficiently well known to operators; these cases have been described, particularly when success has crowned surgical effort, by myself and others in previous communications and addresses. Despite 'territorial' differences in the ulcer story, and in tribute to a great surgeon of a great Allied Power, here are appended accounts of two of Judin's own cases communicated to me; they have indeed been rescued from the jaws of death by courageous surgery.

CASE REPORT

Case 1.—A boy of 17, a waif, was making his way to Moscow from Kiev on the steps and buffers of trains. This journey in frosty weather lasted five days, during which time he got practically nothing to eat. At the station in Moscow the boy ate a kilogramme of brown bread straightaway, and was picked up there in a pool of bloody vomit. An ambulance brought him to Judin's clinic on Feb. 25, 1933. Emaciated in the extreme; physically under-developed; looked 15; waxy pale; cyanotic. Pulse very weak and fast. Hæmoglobin 21 per cent.

The patient's faintness and emaciation, as well as his age, which seemed to negative the possibility of an ulcer, caused Judin to postpone operation.

At daybreak on Feb. 26 another hæmatemesis. 300 c.c. cadaver blood transfused. That day passed uneventfully, but in the night of Feb. 27 another formidable collapse occurred: complete loss of consciousness, no pulse, pupils dilated. Anus paralysed, and spontaneous evacuations of liquid, tar-like stools. The almost-dead patient was given 150 c.c. more of cadaver blood. Pulse and consciousness returned.

At daybreak on March 1 there was again vomiting of crimson blood. Breathing was of the Cheyne-Stokes type, pulse hardly to be felt, and only in the carotid artery.

Judin had at his disposal about 1·5 litres of suitable blood. They gave him 330 c.c. and put the half-dead boy on the operation table. Laparotomy with hardly any anæsthesia. An enormous duodenal ulcer discovered penetrating into the pancreas; erosion of the gastroduodenal artery. A rather difficult resection performed according to the Polya-Finsterer method. A further 1100 c.c. cadaver blood transfused, this being a large quantity for a child. The pulse became excellent; cyanosis of lips and fingers disappeared. On March 3 patient quite fit. Hæmoglobin 27·7 per cent; erythrocytes 2,100,000. Was fed for the first time after nine days' starvation. On April 1 hæmoglobin 47 per cent; erythrocytes 2,780,000.

Discharged on April 24 in a thriving condition with hæmoglobin 64 per cent and 4,240,000 erythrocytes.

Case 2.—One of the head engineers of the railway line from Moscow to the Donetz Basin—a man of 34—had for more than two years been suffering from a duodenal ulcer, and had become an inveterate morphiomaniac. On Dec. 10, 1938, a tar-like stool, and profound faintness. On Dec. 23 profuse hæmatemesis and collapse. The special railway car, where this man was working, was uncoupled at a flag station, 150 kilometres from Moscow, and doctors from a near-by hospital were summoned to this. It happened at the very time of the Twenty-fourth Congress of Russian Surgeons in Kharkov, so that it was not an easy job to secure one of the few professors of surgery remaining in Moscow. The patient was transported from the car to the hospital and immediately operated upon. An enormous duodenal ulcer was found penetrating deep into the pancreas. In view of the patient's grave condition and the ulcer being inaccessible for resection, a ligature of the afferent vessels was performed.

During the night following the operation, the hæmatemesis recurred, and the next day the patient lost so much blood by repeated vomiting that his condition was considered hopeless. A few days later he was transported to Moscow by railway.

On Jan. 2, on Judin's return from the Congress, he saw the patient in consultation. It was the ninth day after the first operation: the hæmoglobin was 18 per cent; erythrocytes 1,500,000. Hæmatemesis had ceased, although the stools continued black. There were no clear indications for a second operation.

On Jan. 10 another intense hæmatemesis and profound collapse. The patient had now but 11 per cent of hæmoglobin left, and only 850,000 erythrocytes. With all possible precautions the patient was transported from Toushino to the Sklifassovsky Institute—about 10 miles.

At operation by Judin, the infiltration around the enormous ulcerous crater made it impossible to locate exactly the previous ligatures on the upper and lower borders of the duodenum. The anterior wall of the duodenum was opened in a V-shaped manner. An enormous crater was discovered, in the centre of which both ends of a big eroded artery with protruding thrombi could be seen; in smearing the cavity of the ulcer with iodine Judin took care not to touch these thrombi. The duodenum was sutured like a cone, which permitted it to be folded in three twists like a "snail". With this "snail" Judin succeeded in tightly plugging the crater, fixing the stump with an additional row of sutures. The operation terminated in a wide gastric resection.

On the operation table the patient was given 1250 c.c. blood, and when returned to bed an additional drip-method of transfusion was instituted. He thus received 2 more litres of cadaver blood in the course of twenty-four hours: a litre from one corpse, stored for six days, and a litre of eight days' standing from another corpse.

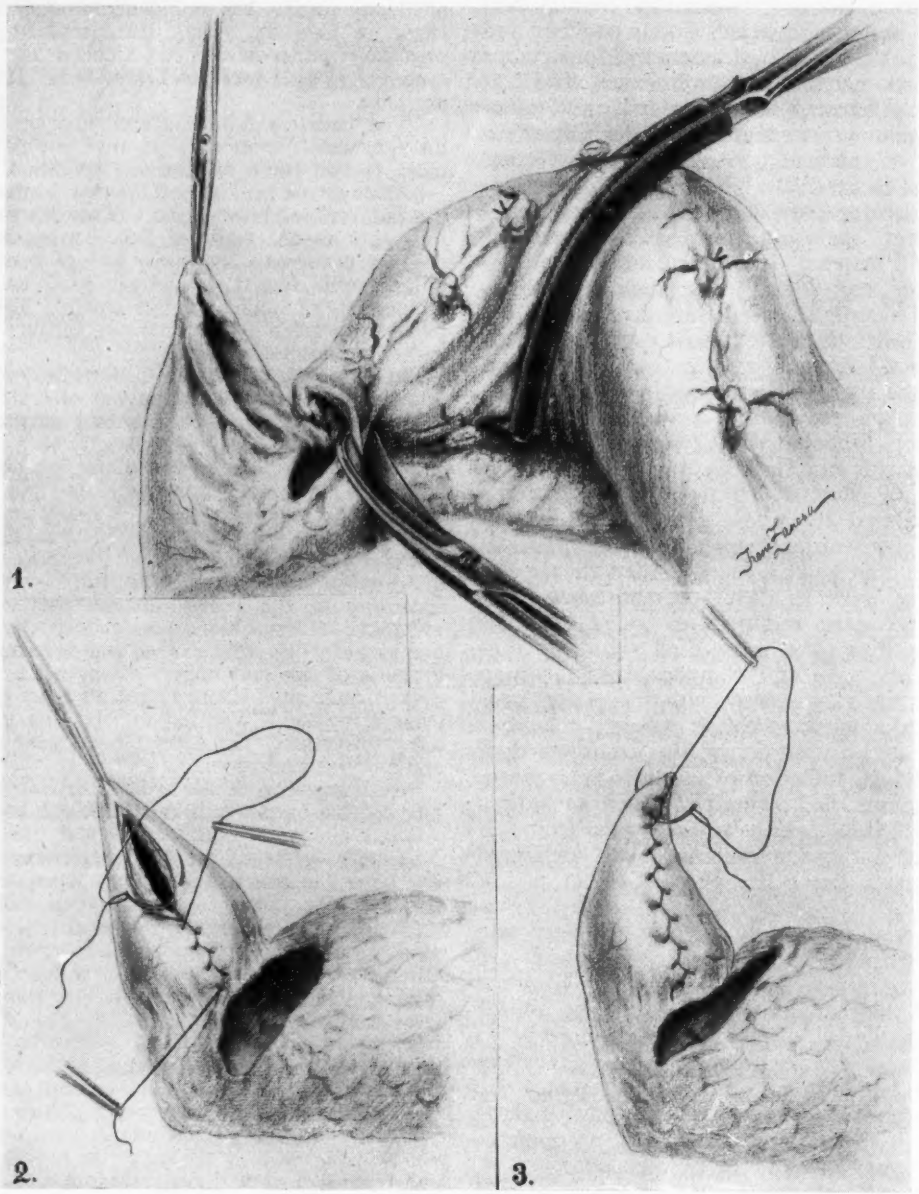

FIG. 374.—Judin's technique. Indicating the operation steps freeing the first part of the duodenum from the base of the penetrating ulcer and its conversion into a conical form.

These 3½ litres of blood quickly effected a change in the patient's appearance and general condition. The hæmoglobin rose from 11 to 46 per cent, and later to 57 per cent. He has remained well ever since, steadily putting on weight and working hard.

HÆMORRHAGE AND PERFORATION

In previous communications I have written at some length on my own experience of the association of perforation and hæmorrhage occurring synchronously or successively, and expressed my own view

that it was probably in the best interests of the patient that these twin complications of peptic ulcer should happen together (!) Nothing has emerged from the untoward experiences of other surgeons to dissipate the dismal prognosis attaching

At Middlesex Hospital, H. L. McMullen had recently under his care a patient who within a few weeks suffered two hæmorrhages and two perforations of a duodenal ulcer. Admitted as an in-patient for severe hæmatemesis he perforated ten

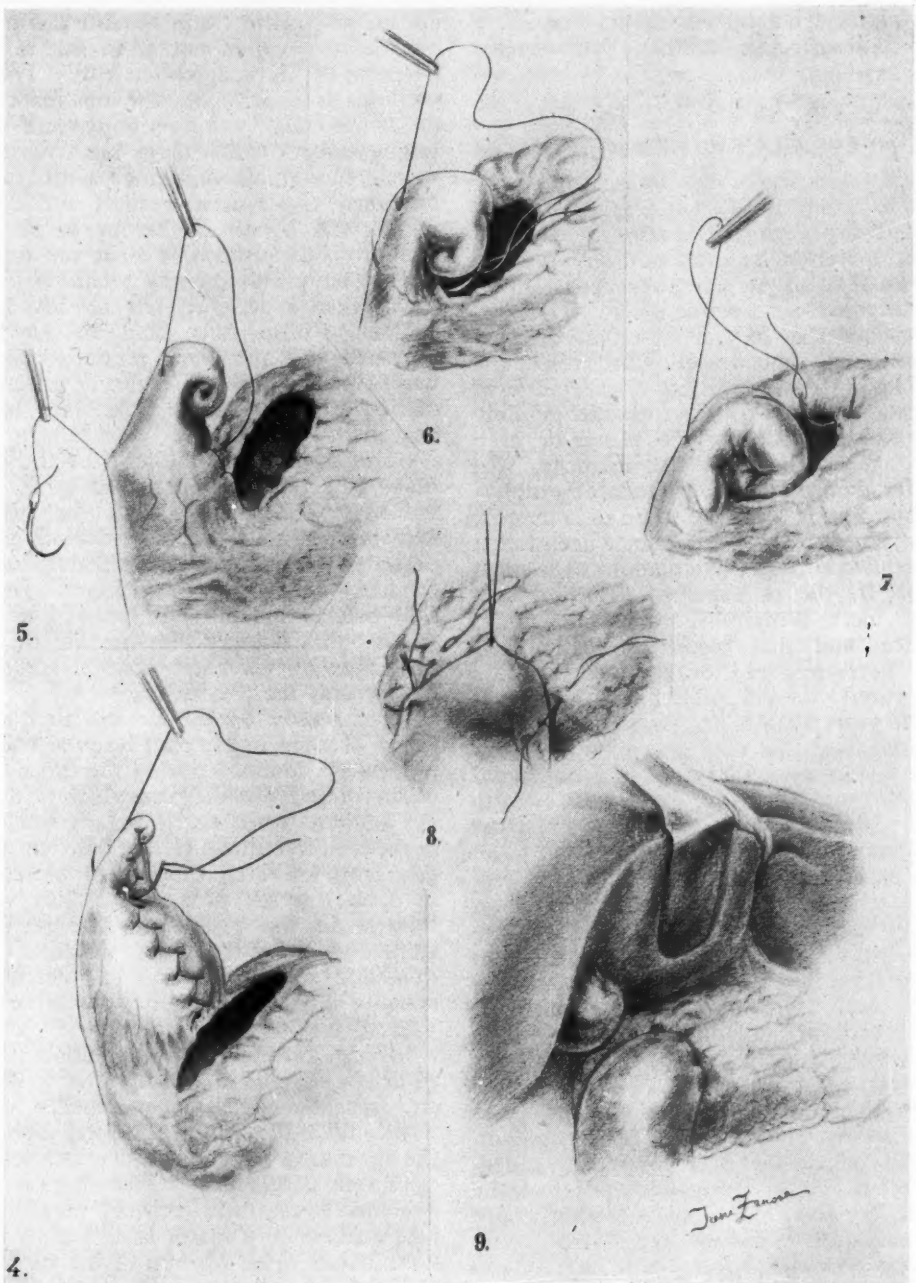

FIG. 375.—Representing the conversion of the cone into a snail-like form, and the employment of this to tampon the ulcer base.

to those patients who bleed and then perforate or perforate and subsequently suffer gastric hæmorrhage. For example, Mr. Ebrill, of St. George's Hospital, satisfactorily sutured a perforation of a duodenal ulcer; a fortnight later a severe hæmatemesis occurred, from which the patient died in four days.

days later; the perforation was successfully sutured. A fortnight later another hæmorrhage occurred, and during the period of recovery from this a second perforation took place from which the patient succumbed. A history such as this lends some support to the practice of primary gastroduodenal resection in the treatment of perforated peptic ulcer as

advocated by Professor S. S. Yudin, of Moscow, in his well-organized Emergency Hospital (Sklifassovsky Institute) with its excellent first-aid and wonderful transport facilities. McMullen's case is, of course, fortunately exceptional, and were primary gastrectomy to become the approved method of treatment for perforated peptic ulcer, doubtless the mortality-rate of this frequent emergency would assume a much higher figure in this country than obtains to-day.

THE ROLE OF SURGERY IN THE PREVENTION OF ULCER HÆMORRHAGE

In a previous number of the BRITISH JOURNAL OF SURGERY I have discussed such cognate subjects as the prospective protection afforded by surgical operation against hæmorrhage from ulcer, and the prospective mortality of an adequate operation to prevent hæmorrhage from a peptic ulcer. The conclusion was reached that in very experienced, competent hands an operation of resection or " resection-for-exclusion " (Finsterer) carried a mortality of about 3 per cent—a figure as great as, and possibly greater than, the risk run by the possessor of a peptic ulcer of death from hæmorrhage. An operation undertaken solely on grounds of prophylaxis against bleeding is therefore to be discouraged.

In the past, surgeons have sometimes been lulled into uneasy acquiescence in the situation, or a spirit of pious hope, by the oft-repeated assertion that fatal cases of ulcer hæmorrhage never occur in private practice, and that blood transfusion has scarcely ever been required or enjoined for this seemingly favoured class of sufferer (!) Nevertheless, during the years 1924 to 1933 many of my own fatal cases belonged to that very group whose future is alleged to be so rosy. Few physicians would venture to make such a fantastic claim to-day, or to assert that blood transfusion as at present employed probably costs more lives than it saves.

The time may come when medical science will of a certainty cure every ulcer of the stomach or duodenum, but until that golden era ulcer hæmorrhage, severe ulcer hæmorrhage, will continue to occur and recur, gravely menacing life and perchance ending disastrously. The problem of the bleeding peptic ulcer is certainly not one of surgery versus medicine, although without a doubt there are cases which operation alone may save. That a predominant percentage of ulcer hæmorrhages cease spontaneously or by reason of medical treatment, to which blood transfusion may have been added, is not denied; it is also freely admitted that the success which has followed early operation might well have attended an expectant line of treatment; but it is no less true that late surgical intervention consequent upon unsuccessful medical therapy is associated with mortality; therein lies the dilemma in treatment.

I am merely pleading once again that the surgeon be not excluded from the consultation until that last desperate stage of ulcer hæmorrhage, the anxieties of which neither physician nor experienced operator is anxious to shoulder. *Every case is to be regarded as a special problem deserving of the most careful attention.* Supreme judgement and self-restraint on the part of the surgeon will be of more value to his patient than the acme of technical skill; indiscriminate operation cannot be too strongly condemned. The value of the continuous blood drip transfusion has proved inestimable, and is nowadays attested by all.

The pioneer is rarely the exploiter, and the man who would iconoclastically destroy an old edifice of worthy tradition and mark out the lines of a new one does not often live to see the walls begotten of his imagination rise. The caution and wisdom of experience, the obstinate conservatism of critics who would rightly and intransigently retain features which usage has triumphantly vindicated, the frank and ingenuous interchange of opinions, the mutual respect for differing viewpoints, the honest endeavour to attain agreement and promote advance, will in the fullness of time erect a temple of therapy retaining in its structure much that experience has already hallowed, but presenting also new bold bastions, the daring minarets and the stark pinnacles that shall bear witness to the devoted service of priests and acolytes carried in consummate perfection to the highest heavens.

The advocacy of surgery for the acute hæmorrhage of peptic ulcer practised by Finsterer, Judin, the writer, and others since the last World War and promulgated specially during the years that preceded the recent Armageddon, drew on itself the fierce light of hostile criticism. But, as has been said before, the insolent innovation has produced at least this salutary repercussion that it made the physicians survey their position and practice more closely and more critically.

The steady decline in mortality of this special group of cases in my own hospital from that dismal percentage obtained during the decade 1924 to 1933 to the present small figure which at the outbreak of the present world conflict characterized the results of medical treatment, clearly betokens the wisdom of the segregation of the severe ulcer hæmorrhages in special wards under physicians with a special interest in the problem. The results emanating from the other hospitals which I have already quoted proclaim with no less certitude the great advance that medicine has made in the past ten years.

If in certain cases operation should appear probable or possible, it may be well for physician and surgeon alike to remember a critical " third day ", since Finsterer's first forty-eight hours is still the optimum period for surgical attack in the bleeding of chronic peptic ulcer. Nevertheless, there is much evidence to-day that recovery may follow operative intervention at a much later period than that just mentioned. The wisdom of the physicians and the skilful hands of many surgical friends have rescued even ' late ' cases on the very brink of the grave.

SUMMARY

1. Profoundly depressed by the fatality of ulcer hæmorrhage treated by medical measures in the early period after the last war I commenced to operate, where possible, on these cases, operation being associated with the transfusion of blood. Most were early operations; the mortality was 9 per cent.

2. After some years and for various reasons my own surgical zeal in regard to these cases waned, but was stimulated once again by the disturbing mortality-rate of 24 per cent attendant on cases of hæmorrhage of proved ulcer origin treated in the medical wards of Middlesex Hospital from 1924 to 1933. This figure coincided very closely with that reported from several other large hospitals.

It was further ascertained that a huge majority of patients with serious ulcer hæmorrhage seen by me in private practice between 1926 and 1933 and not operated on had succumbed; whereas 8 out of the only 10 operated on by me during that period survived, the 2 cases fatal being 'forlorn hopes'.

3. From 1933 to 1939 I again began to employ surgery and operated on nearly 20 per cent of the ulcer hæmorrhage cases coming under my notice. Of 18 cases operated upon within twenty-four hours of the onset of the bleeding only 1 died—a mortality-rate of 5 per cent. Of 11 cases operated upon 'late', 5 died. This contrast in fatality-rate between early and late intervention seemed strongly to support the experience of Finsterer, Arthur Allen, of Boston, U.S.A., and others.

4. The impertinent promulgation of a surgical doctrine for a malady regarded from time immemorial by the physicians as peculiarly their own re-awakened indignant medical interest, especially on questions of transfusion, prevention of dehydration, and early feeding, etc.

5. The results of the non-operative treatment of ulcer hæmorrhage have improved enormously in the past seven or eight years. The mortality now is only about 10 or 11 per cent, sometimes far less, despite the aged and infirm patients met with in many institutions.

6. There are even laudable signs of some dissatisfaction to-day among certain physicians concerning the results of medical treatment, including transfusion, in the more fatal chronic gastric (and perhaps the duodenal) ulcer hæmorrhages in males over 50, and there is an evident disposition to invite and seek surgical aid for these cases.

7. The very best results in the treatment of ulcer hæmorrhage appear to be forthcoming in those hospitals where co-operation between physician and surgeon is closest and the team-spirit is most evident.

8. Blood transfusion given at the rate of about 40 drops a minute is perhaps the most valuable single factor in the saving of life. The debt of medicine and surgery to the Marriott-Kekwick "drip transfusion" is immense.

9. The influence of age upon mortality has been demonstrated by many observers. Comparisons of the statistics of individuals, institutions, or of methods of therapy are worthless which do not take this factor into the reckoning.

10. My own contribution to the subject has been largely that of an irritant or provocative, re-awakening the interest of the physicians in their own subject, wherein the most valuable advances have been made in the past few years and the fatality of the condition notably lowered by their efforts.

In my anxiety to paint a true picture portraying the present position of surgery in relation to the hæmorrhage of gastric and duodenal ulcer, I have not hesitated to solicit the advice and help of numerous interested physicians and surgeons. For permission to avail myself of statistics emanating from institutions under their control I gladly acknowledge the kind co-operation of Surgeon Vice-Admiral Sir Sheldon Dudley, K.C.B., F.R.S., Medical Director-General, Royal Navy; Sir Allen Daley, Chief Medical Officer to the Hospitals of the London County Council; and Dr. H. N. Cameron Macaulay, Chief Medical Officer to the Hospitals of the Middlesex County Council. To my many friends who have aided me so nobly I have already tried to express my indebtedness and gratitude in an earlier portion of the address; no less than the surgeons, the physicians have indeed proved themselves in this inquiry a loyal "band of brothers".

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A MICRO-INCINERATION STUDY OF THE FLAT EPITHELIAL LAYER COVERING THE ALIMENTARY TRACT

By FREDERIC DURAN-JORDA

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THE gastric and intestinal mucosæ are covered by a layer of flat epithelial cells which contains a variable number of diapedesic cells and a network of capillaries (Duran-Jorda, 1945). Between the layer and the pits of the glands there is a kind of albuminous fluid which in some stomachs becomes clotted and produces a cast of the whole elements contained in the flat epithelial layer. This layer is very friable, and more so because it is bathed in the juices contained in the gastric and intestinal mucosæ. The link between the layer and these mucous membranes is very fragile, and it was for this reason that we were compelled to develop a new method of fixation

Fig. 376.

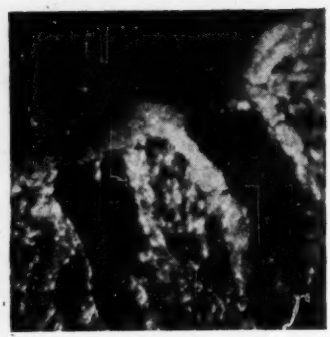


Fig. 377.

FIG. 376.—Gastric mucosa showing two layers (f.v.f., hæmalum and eosin).

FIG. 377.—Micro-incineration of *Fig.* 376 in which only one layer can be seen. The one at the top was mucous material which has disappeared after incineration.

to study the layer. This method will be referred to herein as "formalin vapour fixation" (f.v.f.), and the fixation was carried out by submitting the tissues to formalin vapour in a closed atmosphere, at room temperature, for two to three days.

After its fixation the mucosa is ready to be dehydrated and passed through different alcohols to be finally blocked in paraffin wax. We have found this method very satisfactory for studying the nature of the flat epithelial layer, and it is owing to the lack of a suitable method of fixation that this structure has been overlooked by histologists, who have been using liquid fixatives which show only occasional pieces of the covering layer in the gastric and intestinal mucosæ. These portions have been mistaken for cellular elements deposited by accident on the top of the mucous membrane. The destruction of the covering layer can be better appreciated when one studies the photomicrographs published by numerous authors in regard to the different pathological processes of the gastric and intestinal mucosæ, in which can be seen portions of the mucous membrane still covered by remnants of this flat epithelial layer described by us.

As we have previously remarked, the presence of different cellular elements at the top of the mucous membrane is a well-known fact amongst histologists, and statements have been made to the effect that the material present on the top of the undamaged mucous membrane is clotted fibrin containing diapedesic cells (Nedzel, 1943), but its real nature and physiological, protective function have never been interpreted. The material has always been dismissed as unimportant, and as being formed by desquamative cells, wandering cells, cellular remnants from the upper organs, and food debris, which are all included in a supporting background of mucus. It seems paradoxical then, that the supporting material does not stain with mucus stains. Using hæmalum and eosin, the supporting material, unlike mucin, does not take the basic stain but instead stains with the eosin. And if the gastric mucosa is stained by thionin, the covering layer does not take the stain at all. Occasionally, in the colon, the cytoplasm of a very small number of the mucous-membrane cells does stain with thionin, but not the whole mass of the covering layer as should be supposed. Some authors are very emphatic when describing the methods of mucin staining that it is very difficult to demonstrate the presence of mucin cells in the stomach and intestines, and have arrived at the conclusion that "The mucus in the stomach does not stain by any method unless freshly produced" (Mallory, 1942; Bolles Lee, 1937).

As a consequence of these uncertain methods of staining both the cytoplasmatic cellular mucus and the supposed mucus covering the mucous membrane, we decided to resort to a more drastic method of investigation, and after a careful consideration of the problem we concluded that the most satisfactory technique to reveal the organic nature of the flat epithelial layer described by us was to submit the tissues to micro-incineration. Then we could make a subsequent study of the remaining ashes.

Mucin is a compound protein consisting of a protein radical and a nitrogen-containing carbohydrate, glucosamin (Wells, 1918), and as there are no salts in its composition the mucin cannot leave any mineral ashes after incineration. This is a well-known fact by research workers who have used the micro-incineration method of studying the ashes contained in the tissues (Policard, 1923, 1929), and as the mucin does not leave any ashes after its incineration, the spaces occupied in the cytoplasm of the cells secreting mucin or pseudo-mucin are empty when the tissue is studied after incineration, as can be the case with parotid tumours or ovarian cysts. When describing the cells in the mucous membrane of the stomach, small intestine, and colon, micro-incinerists are quite definite that the empty spaces seen in the cytoplasm of some mucous-membrane cells are the places previously occupied

by the mucus, which does not leave any ashes (Scott, 1933). Then we can conclude that if the layer covering the gastric and intestinal mucosæ is basically formed by an accumulation of mucinous material, it will disappear after incineration and be substituted by an empty space (*Figs.* 376, 377).

MICRO-INCINERATION TECHNIQUE

To carry out these studies, we subjected to micro-incineration alternate slides of serial sections of the mucosæ to be investigated. From these sections, the first was stained with hæmalum and eosin, the second was submitted to micro-incineration, and the third was treated with a mucin stain. The specimens selected had previously been fixed by f.v.f. and embedded in paraffin blocks. Occasionally, we incinerated one of the stained slides after taking some photomicrographs of it as a control, and after micro-incineration we took more photographs of the same fields to compare the two.

Our micro-incinerator was a heat-resisting cylinder of 1¼ in. diameter, wrapped with electric resistance wire and protected by an asbestos sheet. This produced just the right temperature, and we found that by raising the temperature slowly we obtained the best results with less shrinking and less disturbance of the tissue and no bending of the glass slide. We timed our incineration accordingly: 2 min. on, 2 min off; 2 min. on, 2 min. off; 3 min. on, 2 min. off; 3 min. on, 2 min. off; 4 min. on—a total of 14 minutes in the incinerator. When the slide had cooled we put a cover slip on and sealed it with paraffin wax, and the observation of the ashes was done using a dark-ground condenser.

FIG. 378.—The junction between the œsophagus and stomach in a rabbit. The layer can be seen passing from one to the other (f.v.f., hæmalum and eosin).

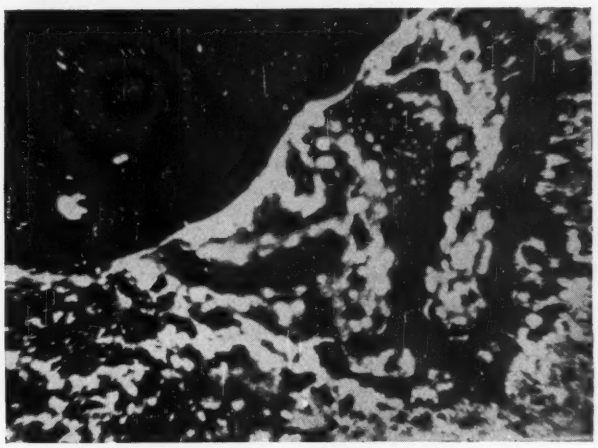


FIG. 379.—Incineration of œsophagus and stomach from serial section of rabbit as *Fig.* 378, showing the layer passing from one structure to the other.

After incineration, the section should first be observed under an ordinary microscope to see if the tissue is still black, in which case the incineration should be longer. If the slide has been well incinerated, the ashes under the ordinary microscope are white and can be seen very faintly.

The specimens of different mucosæ studied came from surgical specimens, as the protective layer is destroyed immediately after death, and we supplemented our investigations by making a further study of some specimens from mammal alimentary tract. These supplementary observations were especially gratifying in studying the connexion between the œsophagus and stomach, which we were not able to

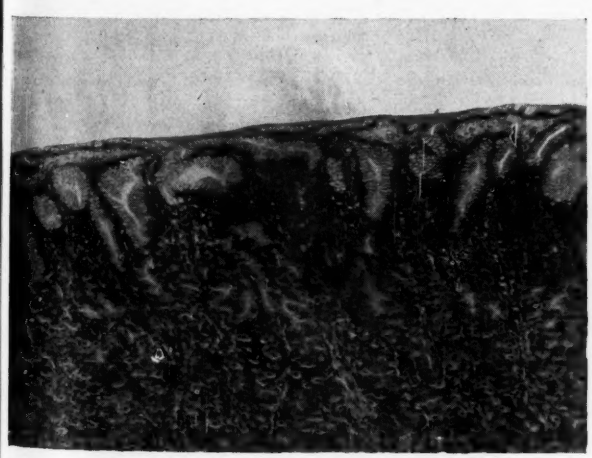


FIG. 380.—Gastric mucosa. Notice the presence of the layer at the top (f.v.f., hæmalum and eosin).

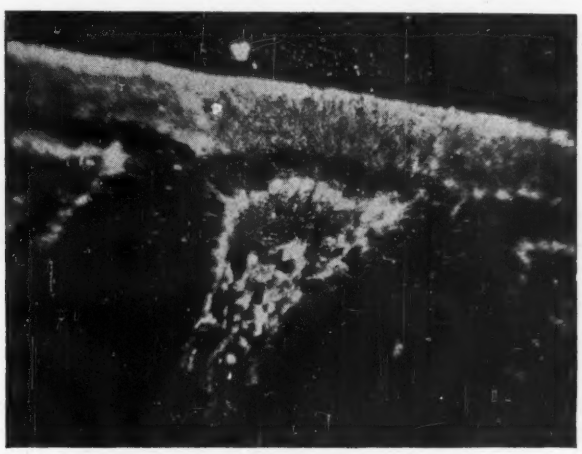


FIG. 381.—Micro-incineration of one field of the previous slide.

investigate in the human gastric mucosa owing to the technical difficulties involved. The animals used for studying the histology of this anatomic region were rabbits, as they are constantly in use in our laboratory for diagnostic purposes.

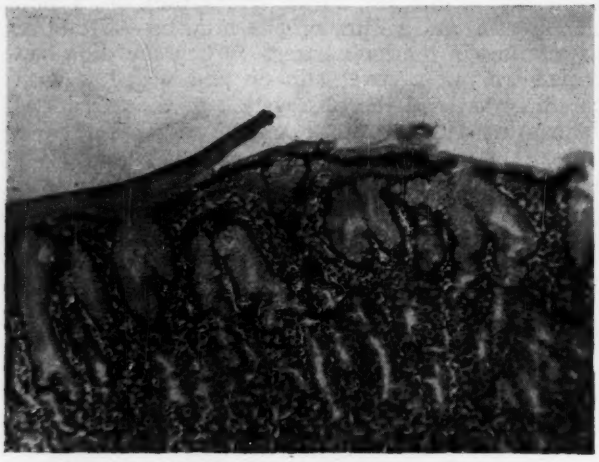


FIG. 382.—Gastric mucosa showing the place where the flat epithelial layer has been removed (f.v.f., hæmalum and eosin).

Stomach.—In the stomach exists a layer which comes from the upper layer of the tunica mucosa of the œsophagus and covers all the stomach, passing to the intestine (*Figs.* 378, 379). This layer has been seen to cover not only the surface of the stomach but also some of the pits of the glands. Sometimes, in the same section, it can be observed how some portions are only formed by a one-cell layer, while other portions are multicellular. This layer is rich in a network of capillaries, varying in size from 7μ diameter, the size of a red blood-corpuscle, to a diameter of 250μ, which is the largest size in our collection. After micro-incineration, the layer leaves thick ashes (*Figs.* 380–383).

Fig. 384.

Fig. 385.

FIG. 384.—Flat epithelial layer which has been removed from the stomach shown in *Fig.* 382 (f.v.f., hæmalum and eosin).

FIG. 385.—Micro-incineration of the layer itself.

The covering layer of the stomach is thicker than the layer found in the small intestine, and the explanation for this could be because of the presence of hydrochloric acid in the gastric mucous. The layer can be peeled off from the stomach if the mucous membrane has been fixed by f.v.f. It is very transparent and can be sectioned. The incineration of the layer itself leaves ashes, demonstrating

FIG. 383.—Micro-incineration of the gastric mucosa where the layer has been removed.

FIGS. 386, 387.—High magnification of the covering layer and its incineration.

that it is not merely dried, inspissated mucous (*Figs.* 384–388).

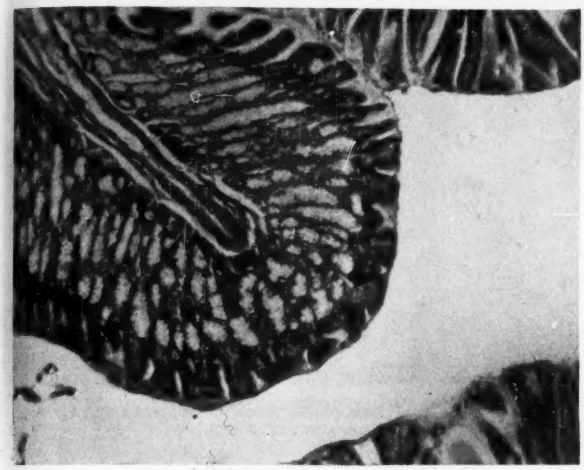
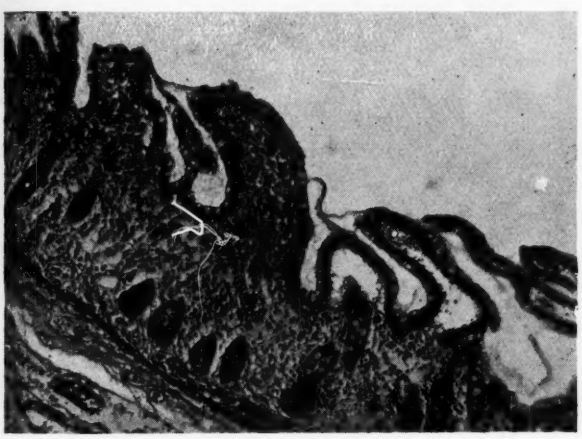
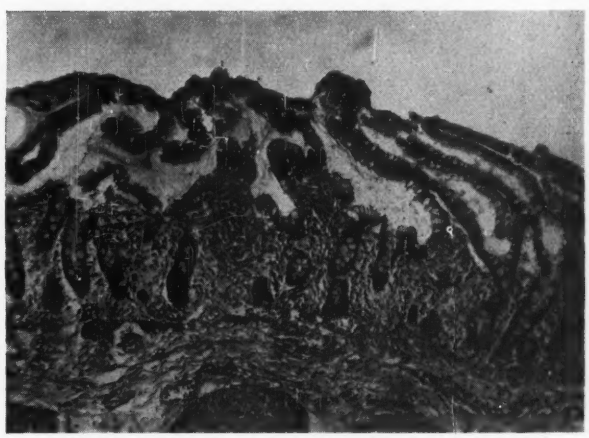


FIG. 388.—Stomach of rabbit showing the whole layer covering the mucosa (f.v.f., hæmalum and eosin).

Small Intestine.—In the small intestine there can also be found a covering layer which is confirmed by incineration. The layer appears to be very thin and contains small capillaries. We have not been able to fine any capillaries as large as the ones present in the gastric mucosa, probably because we have not had enough opportunities for studying the small intestine, as surgical specimens of this organ are scarce. The thinness of the layer can probably be attributed to the fact that in the intestine the *p*H of its media is in the alkaline range (*Figs.* 389–392).

Appendix.—In the appendix the covering layer shows diverse cellular contents and in some microscopical fields is monocellular. In other fields it contains a very large quantity of diapedesic cells. The existence of capillaries can also be demonstrated in this layer covering the appendix. After micro-incineration the ashes are very thick, which is in accordance with the number of cells contained in the covering layer, and the explanation for these thick ashes is quite logical when one considers that the nuclear chromatin of the diapedesic cells is very pyknotic and concentrated (*Figs.* 393–397).





FIGS. 389, 390.—Two sections from normal intestine (f.v.f., hæmalum and eosin.)

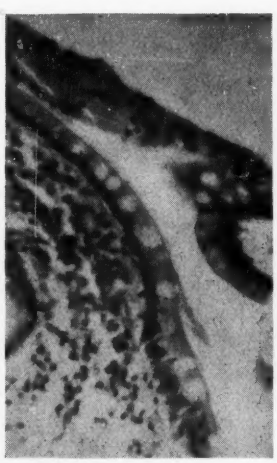


FIG. 391.—Details of *Fig.* 390 under high magnification of intestine (f.v.f., hæmalum and eosin).

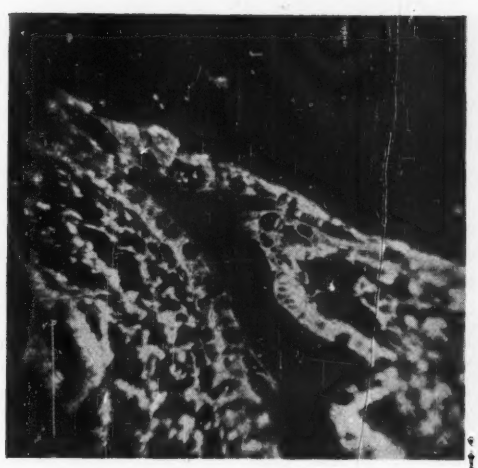


FIG. 392.—Incineration of the same field as in *Fig.* 391.

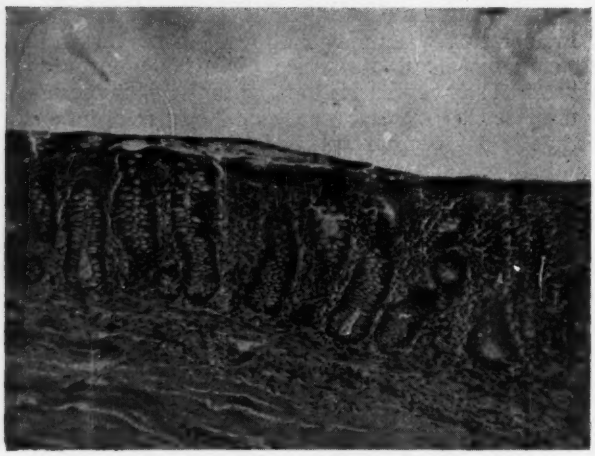


FIG. 393.—Appendix. Showing the covering layer (f.v.f., hæmalum and eosin).

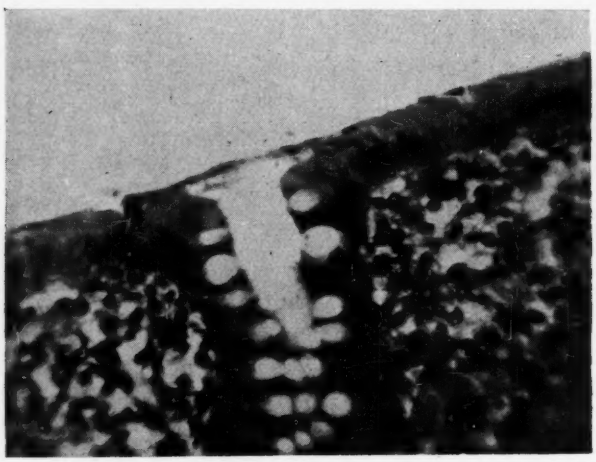


FIG. 394.—High magnification of the appendix showing details of the layer (f.v.f., hæmalum and eosin).

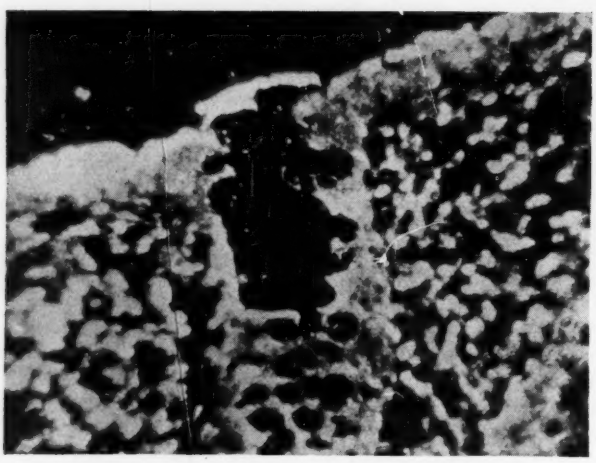


FIG. 395.—The same field as *Fig.* 394 after incineration.

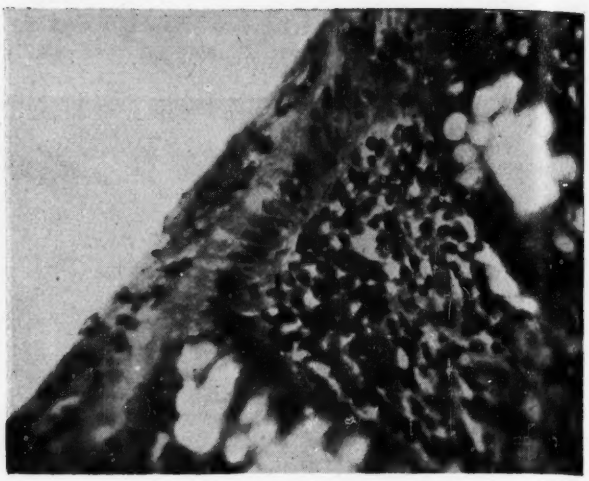


FIG. 396.—A different field from an appendix (f.v.f., hæmalum and eosin).

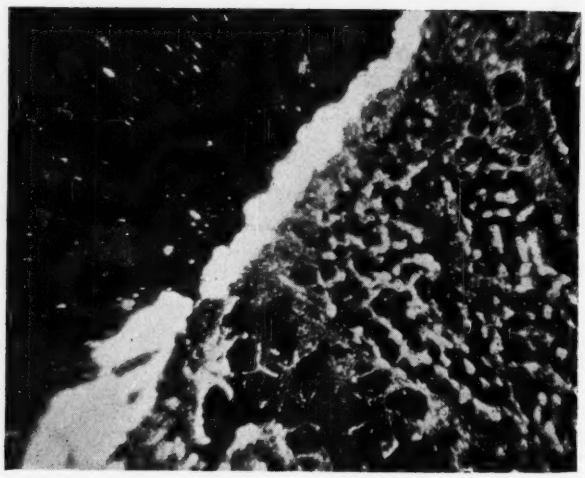


FIG. 397.—The same field as *Fig.* 396 after incineration.

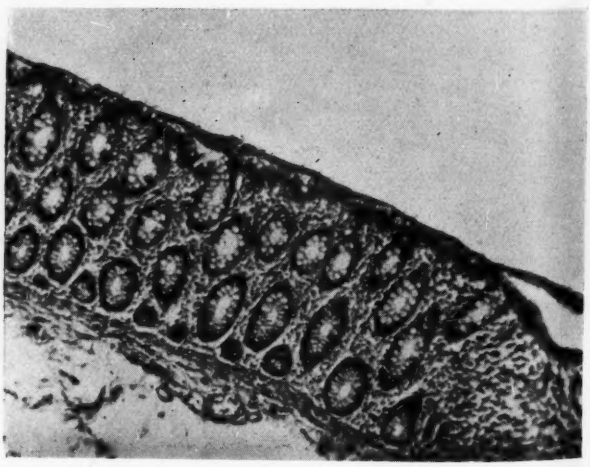
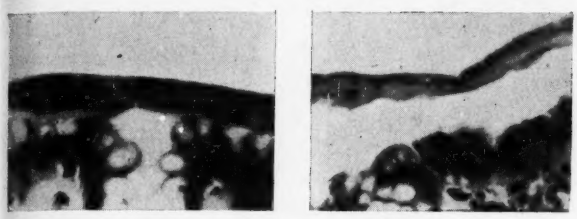
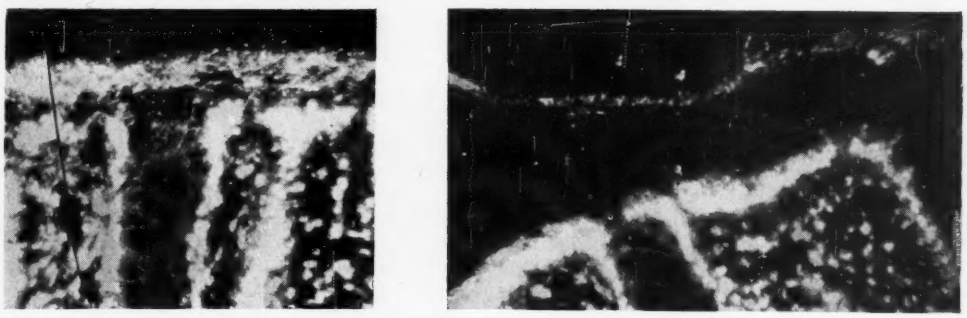


FIG. 398.—Colon, showing the layer all over the surface (f.v.f., hæmalum and eosin).

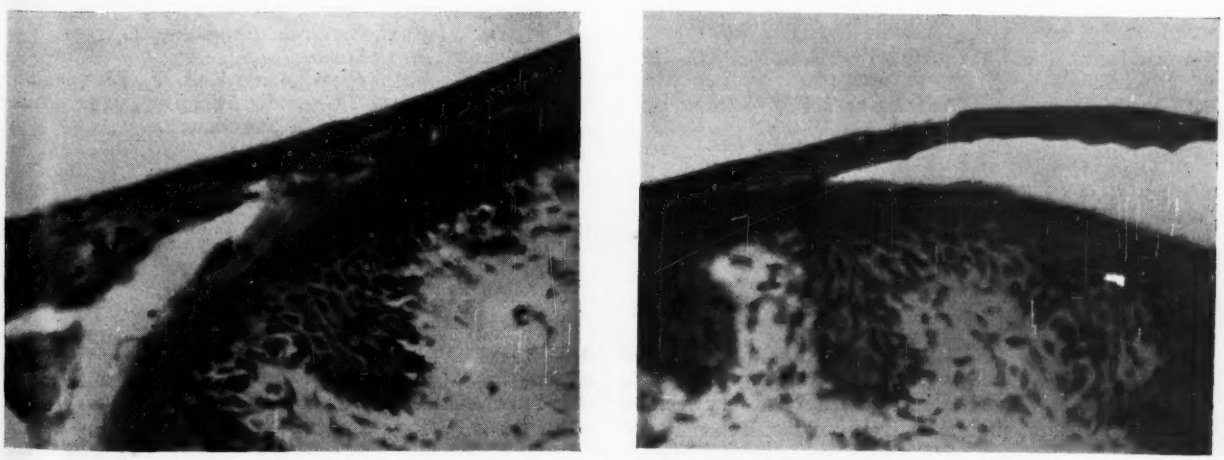
Colon.—In this organ the layer has the same appearance as the layer found in the stomach. Generally speaking, it is very thick and very vascularized, probably due to the fact that a lot of bacterial fermentation takes place in the colon as a result of the anabolism of the hydrocarbonates present in the food remnants, and consequently the mucosa required a thick layer as a protection against the organic acids formed by bacterial and enzymatic fermentation. The ashes of this layer are also very thick, similar to the ones in the gastric mucosa (*Figs.* 398–402).



FIGS. 399, 400.—Details of the flat epithelial layer in the colon. Note the presence of some capillaries (f.v.f., hæmalum and eosin).



FIGS. 401, 402.—Micro-incineration of the fields in *Figs.* 399, 400.



FIGS. 403, 404.—The end of the layer in the stratified tissue at the junction between the rectum and anus. Notice the red blood-corpuscles included in this part of the layer (f.v.f., hæmalum and eosin).

Rectum.—Following the layer from the colon mucosa to the rectum and the anal stratified squamous epithelium, it can be seen how the layer passes from one organ to another and later becomes included in the stratum lucidum of the anal skin. In some of the specimens it is evident that the covering layer still contains capillaries, which are continued in the stratum lucidum of the anal skin, and this raises the question as to whether there are capillaries between the layers of the stratum lucidum and the stratum corneum in the skin, which we have demonstrated to our satisfaction and which will be published elsewhere (*Figs.* 403, 404).

Gall-bladder.—The layer in the gall-bladder appears to be a similar one to that found in the small intestine, and contains a few cells and small capillaries. After incineration it leaves well-marked ashes (*Figs.* 405–407).

After submitting different specimens of mucous membrane from the alimentary tract to the process of micro-incineration, we are now in a position to dismiss definitely the supposition that the existence of this layer is a fake, or is the consequence of the clotting of mucus present in the gastric and intestinal tract as a result of the f.v.f. method of fixation for the preparation of these organs. There can, therefore, be no doubt that the covering layer, which is formed by flat epithelial cells, diapedesic cells, and capillaries, is a genuine organic formation, which should be considered in the future study and description of the histology and pathology of the

FIG. 405.—Gall-bladder—showing the layer (f.v.f., hæmalum and eosin).

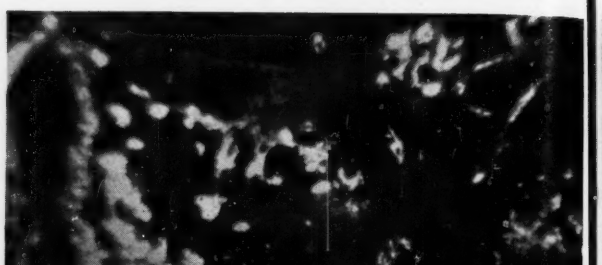


FIG. 406.—Micro-incineration of the same field.

mucous membrane of the stomach, intestines, and gall-bladder.

SUMMARY

The author has demonstrated, by using the micro-incineration technique, the organic nature of the flat epithelial layer, previously described by him, which covers the gastric and intestinal mucosæ and the gall-bladder mucosa.

I would like to thank Professor Wood Jones for his great interest in this work. Also the surgeons of Ancoats Hospital, Mr. P. G. McEvedy, Senior Surgeon, and Mr. J. Philp, Assistant Surgeon, for their willing co-operation in the selection of surgical specimens. I am greatly indebted to my technicians: Mr. C. H. Butcher for his help with the histological sections, and Mr. J. B. Dean, B.Sc., who has done all the photomicrographs. And in addition, I should like to give my sincere thanks to the Lay Board of this hospital who have been kind enough to supply us with the necessary equipment.

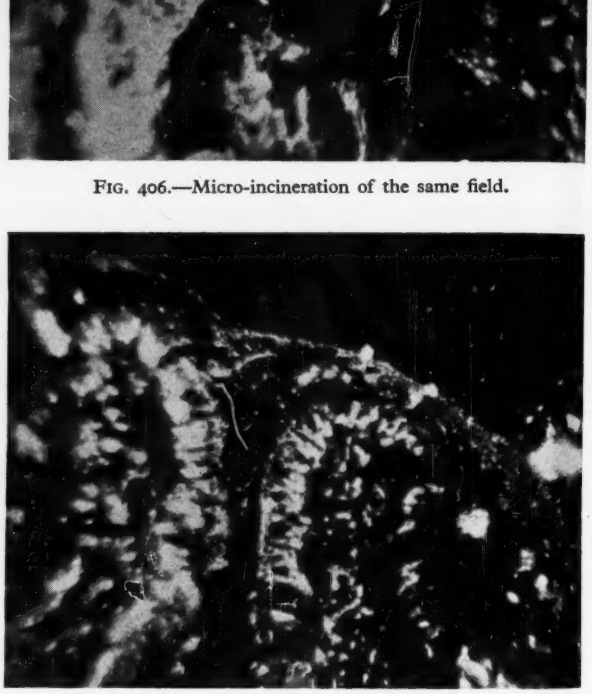


FIG. 407.—Incineration of another field of the gall-bladder where the ashes of the covering layer can be seen.

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METASTATIC OSTEOMYELITIS SECONDARY TO TROPICAL ULCER

BY MARGARET M. SHEPHERD, MAJOR, R.A.M.C., SURGICAL SPECIALIST

IN a series of 98 cases of tropical ulcer in Gurkha soldiers treated in the Combined Military Hospital, Dehra Dun, 11 had metastatic inflammatory lesions in one or more long bones remote from the site of the ulcer. The part of the bone initially and mainly involved, i.e., the cortex of the diaphysis, and the natural course of the disease in the bone, with invariable spontaneous cure, make this a distinctive form of osteomyelitis.

The ulcers were of the type described by Manson-Bahr as tropical sloughing phagedæna, ulcus tropicum, Naga sore, etc. In the series of cases described they occurred on the leg or near the knee. After admission the ulcer passed, clinically, through four stages:—

1. *The Stage of Spread.*—Where the edges are becoming everted and spreading, and the floor is sloughy and purulent.

2. *The Stage of Liquifying Gangrene.*—Here the edges are everted and sometimes 'heaped' and the floor is gangrenous—streaked with alternate blood and pus. Discharge is copious and foul smelling.

3. *The Stage of Commencing Recovery.*—The purulent discharge is thicker and less copious; granulations appear in the floor, and the edges flatten.

4. *The Stage of Epithelialization.*—The floor is of clean granulation tissue, and epithelium grows inwards.

The ulcers were treated in the first three stages by daily or twice daily cleansing by means of gently syringing the ulcer with eusol or boric lotion, and then by the application of gauze soaked in 10 per cent sodium sulphate solution. Elevation of the limb and splinting were sometimes employed. In the healing stage acriflavine and later a dilute ammoniated mercury ointment were used.

In many uncomplicated cases the four stages were achieved in about four weeks, but not infrequently cases occurred in which Stage 2 persisted for six to eight weeks. The long-standing cases often showed evidence of underlying bone reaction, and, in several instances, of bone necrosis and the separation and discharge of sequestra. It is the remote metastatic bone lesion, however, which is of more interest.

CASE REPORTS

In the following case histories technical difficulties, shortage of films, and for one period breakdown of the X-ray apparatus prevented the taking of radiographs as often as was desirable.

Wassermann and Kahn reactions were asked for in all cases. The results are inserted here :—

Case 1. Report not sent.
Case 2. Blood hæmolysed.
Case 3. Negative.
Case 4. Negative.
Case 5. Report not sent.
Case 6. Negative.
Case 7. Contaminated.
Case 8. Negative.
Case 9. Negative.
Case 10. Negative.
Case 11. Negative.

The histories are written in weeks dating from the date of origin of the ulcer as accurately as could be ascertained from the history.

Case 1.—Gokarna Bdr., a Gurkha, aged 21 years.

Admitted Nov. 5, 1943. Ulcer, said to be of fifteen days' duration, was situated about the middle of the left shin and was roughly oval in shape (2 in. × 1 in.), with a dirty sloughing floor and everted edges.

4th week: Complained of pain on the anterior aspect of the right ankle. No fever. On examination, tenderness lower end right tibia. Ulcer clean.

6th week: Ulcer healed. Pain middle of right leg. No fever. On examination, tender swelling of right shin. Radiograph (misplaced) showed linear periostitis and fluffy new-bone formation and decalcification of the underlying cortex about the middle third of the right tibia. White blood-count. Total leucocytes 8600.

10th week: Pain slight. Tender diffuse swelling right shin persists.

14th week: Almost painless. Radiograph showed woolly new bone almost the whole length of the shaft of the tibia, especially marked on the anterior and external surfaces, and some linear periosteal new bone on the proximal part of the inner surface of the shaft.

17th week: Painless. Still slightly tender.

19th week: Very slight tenderness. Radiograph showed increased definition and calcification of the new bone. No absorption. Appearance of the tibia like Paget's disease. (*Fig.* 408.)

20th week: No pain or tenderness.

21st week: No alteration in palpable thickening of right tibia. Discharged to duty.

Case 2.—Jhabi Lal Chettri, a Gurkha, aged 19.

Admitted Dec. 6, 1943. Ulcer, said to be of twenty-one days' duration, was situated about the middle of the left shin and was roughly oval (2½ in. × 1¼ in.), with a dirty gangrenous floor and everted edges. Inguinal adenitis noted. No fever.

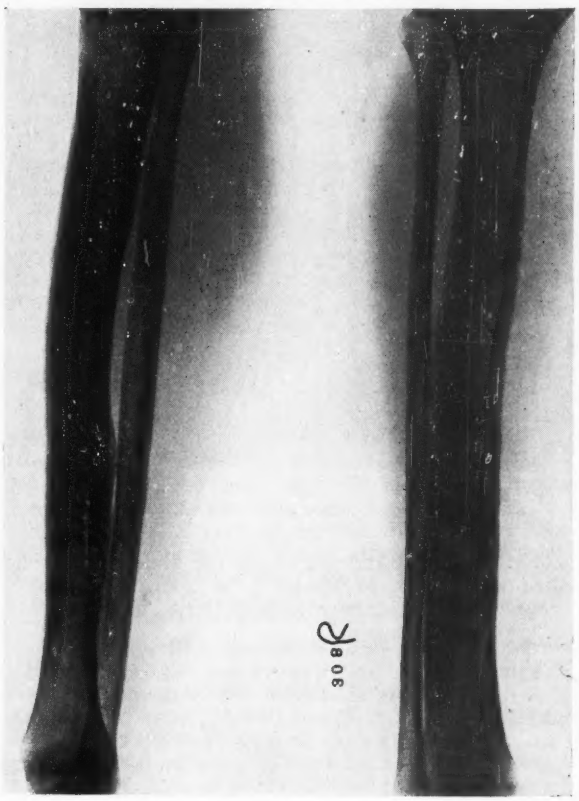


FIG. 408.—*Case* 1. Radiograph taken at nineteenth week.

6th week: Remittent fever up to 100° for four days. Complained of pain in both legs. Vague tenderness. The ulcer was clean. Sulphathiazole 1 g. four-hourly given (up to 30 g.). Some diminution of pain.

7th week: Ulcer clean and healing. A tender swelling was noted in the left tibia distal to the ulcer, and in the lower quarter of the right fibula. On radiography, very slight decalcification in the cortex of the lower end of the shaft of the right fibula and also in the lower end of the shaft of the left tibia.

8th week: Ulcer almost healed. Pain in legs less.

9th week: Ulcer healed. Complained of pain in left thigh. Tenderness lower part of the shaft of left femur. Blood-count—total white cells 10,400; polymorphonuclears 61 per cent, lymphocytes 34 per cent.

10th week: X-ray examination, cortical destruction and fluffy new-bone formation involving the lower third of the shaft of the fibula—the lower limit being 2 in. above the tip of the external malleolus. Similar appearance of left femur, involving about 5 in. of the antero-external surface of the shaft of the bone.

11th week: No pain right fibula, but pain in left femur and left tibia. Fever intermittent up to 100° for two days. Non-tender swelling right fibula. Very tender swelling left lower femur and left tibia.

The pain and tenderness gradually settled in the left tibia, but persisted longer in a mild form in the left femur.

17th week: No pain or tenderness in thighs or legs. Radiograph of right fibula showed increased calcification and definition of the new bone with a flattening of its

outline, though little resorption had taken place. Apparently similar changes in left tibia. Left femur showed much irregular new bone involving the shaft more extensively than on the previous X-ray examination.

FIG. 409.—*Case* 2. Radiograph taken March 9, 1944.

Calcification has taken place and the appearance is not unlike that of melorheostosis. (*Fig.* 409.)

19*th week*: Discharge to light duty.

Case 3.—Tilak Bdr., a Gurkha, aged 19 years.

Admitted Dec. 21, 1943. Ulcer, which was said to be of two months' duration, was situated about the middle of the right shin, was about 1¼ in. × 1½ in. in size. It had a moderately dirty floor and everted edges.

9*th week*: Complained of pain in both forearms, particularly in the right. On examination, no fever. Slight tenderness, middle right radius.

11*th week*: Slight pain persists in the right forearm. On examination, diffuse tender swelling in right radius in the middle and lower third.

12*th week*: Ulcer clean.

14*th week*: Ulcer healed. X-ray examination showed osteomyelitis of the middle half of the right radius, with bone destruction and both fluffy and linear periosteal new bone (*Fig.* 410). White blood-count—total leucocytes 10,800; polymorphonuclears 72 per cent.

18*th week*: Pain very slight and tenderness less. Palpable thickening of both radii.

20*th week*: No pain or tenderness. Radiograph showed some little absorption of new bone in right radius, consolidation of the remainder. Some decalcification persists (*Fig.* 411). Left radius showed a similar appearance, although less marked.

21*st week*: Discharged to duty.

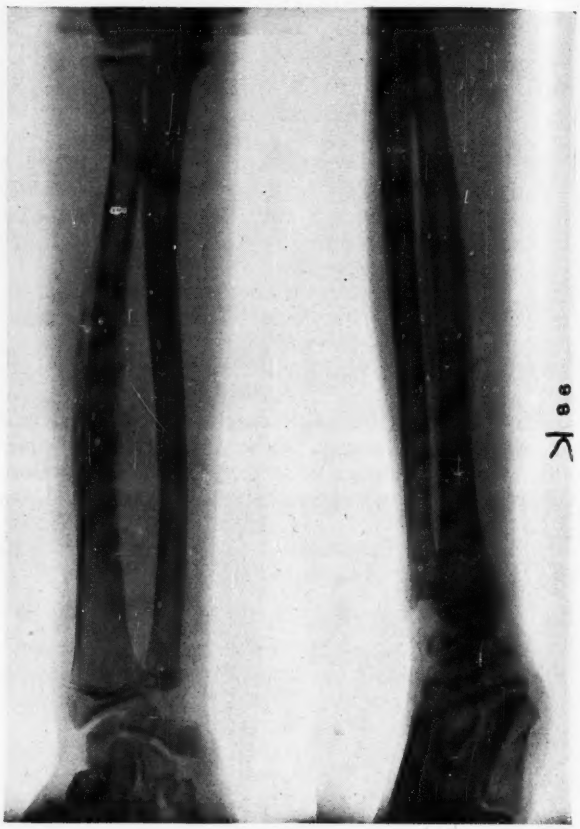


FIG. 410.—*Case* 3. Radiograph taken Jan. 22, 1944.

Case 4.—Bhim Bdr., a Gurkha, aged 35 years.

Admitted Aug. 31, 1943, with an infected laceration in right infrapatellar region.

3*rd week*: Noted as a tropical ulcer, 2½ in. × 2½ in.

About the fifth week complained of pain in both legs and in the left clavicle.

10*th week* (first seen by writer): General condition poor. Pain in legs and left clavicle. On examination, tender thickening of middle left clavicle and middle of both tibiæ. No fever. X-ray examination, left clavicle showed

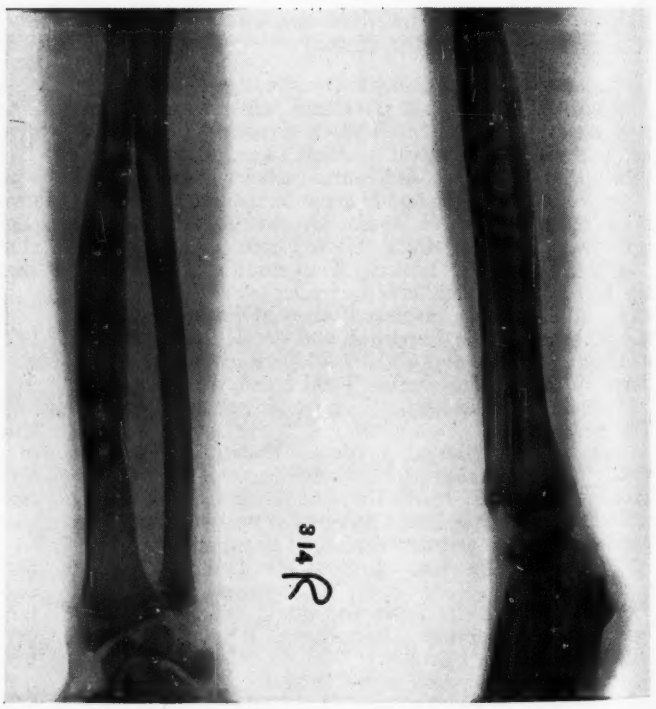


FIG. 411.—*Case* 3. Radiograph taken March 9, 1944.

decalcification and infective erosion of the outer half. A little faint new-bone formation. Left tibia showed decalcification suggesting osteomyelitis of the lower two-thirds of the shaft. A little faint subperiosteal new bone. Right tibia showed similar changes in the middle and the upper thirds of the shaft.

11*th week*: Fever up to 101°. Blood-culture sterile.

13*th week*: Fever continues up to 102° at night. Sulphathiazole 1 g. four-hourly.

14*th week*: 24 g. sulphathiazole given. Clavicle less painful and less tender.

15*th week*: Afebrile. Shins less painful and tibiæ less tender.

16*th week*: Still afebrile, but pain and tenderness in the upper part of the shafts of both tibiæ have increased.

20*th week*: On radiography, left tibia showed increased bone destruction throughout the whole thickness of the upper part of the shaft; some periosteal new bone. Right tibia gave a similar appearance. Leucocyte count, 15,000; polymorphonuclears, 76 per cent.

21*st week*: No pain. Thickening of tibiæ persists. No tenderness. General condition has improved.

22*nd week*: Right foot-drop developed, with blunting of sensation in the right anterior tibial and musculocutaneous areas of sensory supply. Slight œdema both ankles.

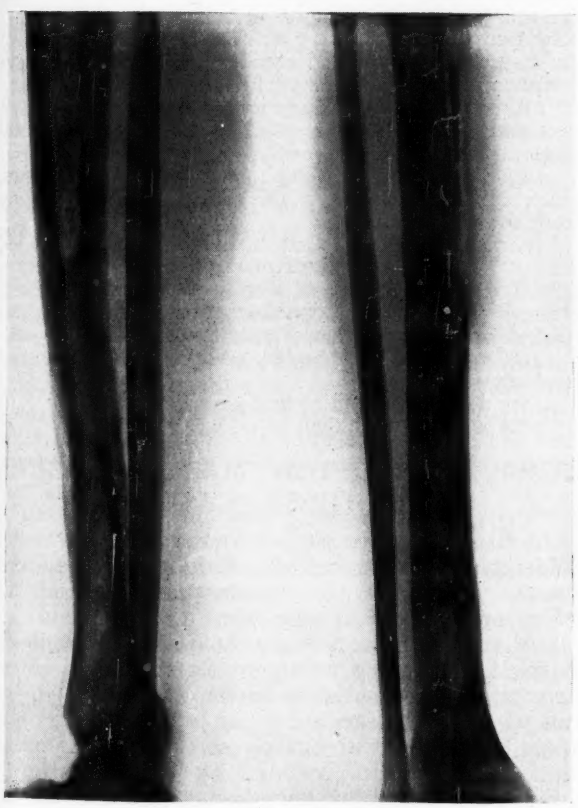


FIG. 412.—*Case* 4. Radiograph taken Feb. 27, 1944.

23*rd week*: Medical board held. Recommended discharge from the Army as unfit for further service.

25*th week*: General condition greatly improved, still has slight pain and tenderness in both shins. X-ray examination: Right tibia, osteomyelitis of the upper part of the shaft, with fluffy periosteal new bone, and denser areas of old cortex, which appear to be sequestra; in the lower part of the shaft there is decalcification, erosion of cortex, and periosteal new bone. Left tibia, very similar changes (*Fig.* 512). Clavicle, dense bone.

27*th week*: Pain and tenderness in the right shin and the slight œdema persist. Very slight pain and tenderness in left shin.

28*th week*: No pain. Slight tenderness right tibia. No œdema.

29*th week*: No pain or tenderness.

31*st week*: Discharged from hospital. Apart from recovering foot-drop, fit for work.

Case 5.—Chettri Bdr., a Gurkha, aged 17 years.

Admitted Nov. 4, 1943, with multiple small ulcers on the outer side of the right leg, said to be of one month's duration. The total ulcerated area was 3 in. × 3 in. and the ulcers had sloughy floors and everted edges.

6*th week*: Complained of pain in the front of the right thigh. No fever. Ulcers have coalesced, but are clean and epithelialization has begun.

8*th week*: Pain persists. Tender thickening of the lower part of the femoral shaft. X-ray examination showed linear and faint fluffy periosteal new bone around the whole thickness of the lower half of the shaft of the right femur; slight decalcification in the cortex on the anterior aspect of the lower shaft.

11*th week*: Ulcers healed. Pain less, but slight intermittent fever up to 99°.

12*th week*: Afebrile. Slight pain and tenderness.

14*th week*: No pain. Slight tenderness right femoral shaft.

18*th week*: No pain. No tenderness. Wishes to be discharged, but detained for X-ray examination, etc. Leucocyte count, 8800.

21*st week*: X-ray examination showed consolidation and calcification of new bone, causing irregular thickening of the lower half of the right femoral shaft.

23*rd week*: Discharged to full duty.

Case 6.—Tanka Bdr. Thapa, a Gurkha, aged 22 years.

Admitted Sept. 13, 1943, with an ulcer, said to be of one month's duration, about the middle of the right shin. The ulcer was 3 in. × 2½ in., with everted edges and a gangrenous floor.

16*th week*: Complained of pain in both thighs. Ulcer clean and healing. Febrile. Evening temperature up to 100°. On examination, tender thickening, both femoral shafts. Leucocyte count, 18,400; polymorphonuclears, 82 per cent.

17*th week*: X-ray examination of right femur showed cortical decalcification and erosion for 7 in. of the lower and middle part of the shaft anteriorly; linear and fluffy periosteal new bone externally, internally, and anteriorly for 5 in. or so. The left femur showed similar, although less marked changes, in the upper half of the shaft on the external, internal, and posterior surfaces.

18*th week*: Irregular fever continues, 99–101° in the evening. Sulphathiazole 1 g. four-hourly (total, 24 g.) given; on completing course the pain was less.

19*th week*: Afebrile. Still some pain in thighs.

21*st week*: Very slight pain. Tenderness persists.

23*rd week*: No pain. Very slight tenderness. Ulcer healed.

24*th week*: Very slight tenderness. Leucocyte count, 7200; polymorphonuclears, 66 per cent.

26*th week*: No pain, no tenderness. Thickening of femoral shaft persists.

27*th week*: Feels quite fit for duty, and appears so. Kept in hospital for a later X-ray examination.

32*nd week*: X-ray examination of right femur showed consolidation and calcification of the new bone with recalcification of the eroded cortex. Left femur showed similar changes, producing irregular thickening of the shaft from half an inch below the lesser trochanter.

33*rd week*: Discharged to full duty.

Case 7.—Prem Bdr., a Gurkha, aged 17 years.

Admitted Dec. 21, 1943. Ulcer, said to be of twenty-five days' duration, situated in the left infrapatellar

region. It was 1 in. × 1¼ in. and had everted edges The floor was fairly clean.

7th week: Complained of pain in the right thigh. Some fever, evening temperature rising to 99°. Tenderness mid-shaft right femur. Ulcer not healing; floor more gangrenous.

8th week: Moderate fever continued till this week. Leucocyte count near the end of febrile period, 6800; polymorphonuclears 75 per cent.

X-ray examination showed linear periosteal new bone on the lateral and anterior aspects of the middle and lower parts of the shaft of the right femur. Faint fluffy new bone on the anterior aspect.

9th week: Ulcer healed. Pain and tenderness persist, but less marked.

13th week: No pain or tenderness.

14th week: X-ray examination showed consolidation of new bone; some cortical decalcification anteriorly mid-shaft.

26th week: Discharged to duty.

Case 8.—Kjs Bdr. Chettri, a Gurkha, aged 17 years.

Admitted Nov. 12, 1943, with two ulcers said to be of thirteen weeks' duration. The ulcers, each 1½ in. × 1 in., were situated on the outer side of the lower third of the left leg. Floors dirty and gangrenous, with some eversion of edges.

24th week: Complained of pain and tenderness of the whole of the inner side of the right shin. No fever. Ulcers cleaning. No definite bone changes on radiography. Leucocyte count, 10,800; polymorphonuclears, 70 per cent.

25th week: Middle third right tibia thickened and tender. Ulcers healing.

28th week: X-ray examination, patchy decalcification and thickening of the cortex of the anterior part of the middle third of the right tibia.

29th week: No pain, no tenderness. Thickening persists.

31st week: Discharged to duty.

Case 9.—Sher Bdr. Ali, a Gurkha, aged 17 years.

Admitted Nov. 15, 1943, with ulcer, said to be of a month's duration, situated on the outer aspect of the lower third of the left leg; it measured 1½ in. × 3 in., and had a dirty gangrenous floor.

13th week: Complained of tenderness right shin. Ulcer becoming cleaner. No fever.

15th week: X-ray examination showed small patch of decalcification in the cortex of the anterior part of the shaft of the right tibia 4½ in. above the lower end. Leucocyte count, 12,800; polymorphonuclears, 65 per cent.

16th week: Ulcer healed. No pain in shin. Slight tenderness. Right tibia thickened and tender at the junction of the lower and middle thirds.

19th week: Two days' fever. Tenderness less.

20th week: On radiography, more extensive decalcification was apparent in the anterior part of the cortex of the lower shaft of the right tibia. Affected area extended for 5 in. Periosteal new bone anteriorly and medially. No pain or tenderness.

22nd week: No pain or tenderness. Discharged to duty.

Case 10.—Tej Bdr., a Gurkha, aged 18 years.

Admitted Nov. 25, 1943, with ulcer, said to be of three weeks' duration, situated superficially to the left internal malleolus. It was 1 in. × 1¼ in. and had everted edges and a dirty floor.

7th week: Complained of pain in left mid shin. No fever. Tender swelling at the junction of the lower and middle thirds of the left tibia.

11th week: Ulcer healed. Shin still tender. X-ray examination: On the anterior surface of the left tibia, at the junction of the lower and middle third, the cortex for 5 in. is decalcified and eroded. Over 7 in. of periosteal new bone, linear and fluffy, is deposited. Between the new bone and eroded cortex is an apparent space.

12th week: Leucocyte count, 13,000; polymorphonuclear leucocytes, 75 per cent.

13th week: Pain slight. Swelling and tenderness persist, but tenderness is decreasing.

16th week: X-ray examination showed recalcification and consolidation of the new bone, with resultant thickening of the shaft of the tibia. The 'space' noted before is still evident.

19th week: Very slight pain and tenderness.

20th week: No pain or tenderness.

21st week: Discharged to duty.

Case 11.—Sahi Bdr., a Gurkha, aged 22 years.

Admitted Nov. 21, 1943, with an ulcer, said to be of two weeks' duration. It was situated on the lower third of the right shin and had everted edges and a gangrenous floor. Mild inguinal adenitis.

6th week: Ulcer still very dirty. Has had intermittent fever, up to 100°, for eleven days.

8th week: Complained of pain in middle of the left shin. No fever now. Tenderness, left mid shin. Ulcer still very dirty.

11th week: Ulcer becoming cleaner. Tender swelling mid tibia (left). Fever intermittent up to 100·2°. X-ray examination: Decalcification of cortex and slight new-bone formation for about 5 in. of the anterior surface of the shaft of the left tibia. Leucocyte count, 18,000; polymorphonuclears, 64 per cent; lymphocytes, 28 per cent.

13th week: Fever continues. Pain and tenderness continue. Sulphathiazole, 1 g. four-hourly, to a total of 20 g.

14th week: Fever settling. Pain and tenderness less. Ulcer still has a slough in the centre, although healing from the edges.

16th week: X-ray examination showed increased destructive changes and decalcification in the cortical bone for almost the whole length of the shaft. These changes, as also new-bone formation, are particularly marked on the external and anterior surfaces.

21st week: Ulcer healed. Very slight pain and tenderness.

26th week: No pain or tenderness.

27th week: Discharged to duty.

SUMMARY OF THE MAIN FEATURES OF THESE CASES

Aetiology.—The patients were all Gurkha males undergoing training in Dehra Dun and the adjacent jungle. The average age was 20 years, the youngest being 17 years old and the eldest 35 years.

All cases developed the bony lesion while in hospital undergoing treatment for the ulcer, hence trauma was not a factor in the aetiology. In 10 out of 11 cases (the exception being *Case* 11) the ulcer was clean and healing at the time of onset of the bone disease, as shown by the occurrence of pain. The onset of bone pain occurred on an average four weeks before complete healing of the ulcer took place.

Clinical Course.—The first symptom of a bone being affected was local pain and tenderness. The pain was not as a rule severe; the patient usually mentioned the pain four or six days after the onset. In two cases only—*Cases* 2 and 4—did severe pain develop. The mildness of pain in some cases may be illustrated by *Case* 3, who complained of pain in the right forearm only and hence radiographs of that limb only were taken. At a

later date thickening of both radii was noticed and X rays showed almost as great an affection of the left as the right radius. Another patient, who attended the out-patient department more recently than these in this series, was sent up because he complained of pain in the left shin during P.T. He had had a small tropical ulcer near the head of the left fibula for which he had not been admitted to hospital. It had healed three weeks before the onset of the bone pain. Clinically the middle of the left tibia was thickened and tender. X rays revealed the typical cortical decalcification and fluffy new-bone formation over the middle third of the inner side of the left tibia.

In two cases—*Cases* 5 and 6—radiographs taken soon after the onset of pain showed marked changes, suggesting that when the bone was first affected there had been no pain. Tenderness persisted after all pain had subsided. Disappearance of tenderness was taken as denoting clinical cure.

In only 3 of the 11 cases, i.e., *Cases* 2, 6, and 7, did fever accompany the onset of symptoms. In *Case* 2 it lasted for four days only at the time of onset, but recurred mildly for a week five weeks later. In *Case* 6 fever was more marked and lasted for two and a half weeks. In *Case* 7 it was mild and persisted for nearly two weeks.

Four cases (*Cases* 1, 3, 8, and 10) ran a completely afebrile course. Of the remainder *Case* 9 had mild fever for two days. *Case* 4 had marked fever for five weeks; *Case* 5 had mild fever for one week; *Case* 11 had moderate fever for four weeks. Sulphathiazole was given in *Case* 4, as he had persistent fever and his general condition was deteriorating. Some relief of pain appeared to result, and the fever was less marked, subsiding the week after the course of sulphathiazole was completed. In *Case* 2, where pain was a feature, sulphathiazole was again tried and again appeared to cause diminution of the severity of the pain. Again, in *Case* 6, fever and pain appeared favourably influenced by the same drug, and the same result was noted in the week following the completion of the course in *Case* 11.

Leucocyte counts were done, but not systematically. A moderate polymorphonuclear leucocytosis was noted in most cases during active bone disease, but the data are insufficient.

Except in *Case* 4, the constitutional upset was mild. No evidence of pyæmia, such as the formation of abscesses, was present. Even when a subcutaneous bone, such as the tibia, was involved there was no reddening of the skin or inflammatory œdema locally. The tissues outside the periosteum appeared unaffected. In no case was sequestrum and sinus formation suggested clinically.

From the first bone symptom to complete clinical healing the average time was 12 weeks—the longest being *Case* 4 (23 weeks), in which a recrudescence of disease in the tibiæ seemed to take place, and the shortest was *Case* 8 (5 weeks), which was the mildest of the series from all aspects. Recovery in the long case (*Case* 4) was as complete as in the others.

All cases, except *Case* 4, which developed an anterior tibial neuritis, with foot-drop, returned to duty. Some were kept in hospital a week or two longer than necessary, and against the men's wishes, for purposes of observation. As no alteration in the local thickening of the bone seemed to be taking place, they were discharged.

Radiologically the first change was a decalcification in the cortex of the diaphysis of a long bone. Then followed linear periosteal new bone deposit and a fluffy or woolly new bone having a broadly crenated outline. In some cases healing then occurred by deposition of calcium salts in the new bone. Very little resorption took place. In other cases changes indicative of osteomyelitis appeared before healing took place. In *Case* 4, where these changes were most marked, parts of the shaft appeared dense and had all the appearance radiologically of sequestra. Yet there was no attempt on the part of the body to extrude these pieces of bone. This patient was kept in hospital four weeks after complete clinical healing had taken place.

THE RELATION OF THE SITE OF THE BONY LESION TO THE SITE OF THE ULCER

Case No.	*Ulcer Site*	*Bone Affected*
1	Left shin	Right tibia
2	Left shin	Right fibula, left femur, left tibia
3	Right shin	Both radii
4	Right infrapatellar region	Left clavicle, both tibiæ
5	Right leg	Right femur
6	Right shin	Both femora
7	Left infrapatellar region	Right femur
8	Left leg	Right tibia
9	Left leg	Right tibia
10	Left internal malleolar region	Left tibia
11	Right shin	Left tibia

I have since seen 2 cases where the shaft of the humerus was involved and so far the only long bones which have not been affected have been the ulna and ribs, but it seems probable that any long bone may be attacked and the commonest are the tibia and then the femur.

The incidence of bone lesions in 11 out of 98 cases is high. At this time all tropical ulcers could not be admitted to hospital owing to shortage of beds, and thus severe or protracted cases were given preference for admission.

CONCLUSION

Eleven cases of inflammatory metastatic bone disease in cases of tropical ulcer have been described.

The bone lesion is unusual in that, although marked radiological changes were present, pain and general constitutional upset were not marked features. The part of the bone primarily involved was invariably the cortex of the diaphysis. In no single case was the metaphysis involved. The peculiar fluffy new bone did not absorb to any extent, and a thickening of the involved bone remained. In no case did any operative procedure appear to be indicated. All healed spontaneously.*

Further study would seem to be indicated.

I wish to thank the radiologist, Major Eates, I.A.M.C., for his help in radiography, and Dr. Sharma, C.M.P., for his great assistance in note taking.

* Since writing the above, a few similar cases have been treated with penicillin and the results are encouraging.

TENDON TRANSPLANTATION FOR RADIAL PARALYSIS*

By R. B. ZACHARY

More than a generation elapsed between the introduction of tendon transplantation for motor paralysis by Nicoladoni (1882) and its extensive use for permanent radial paralysis by Robert Jones (1917), whose technique is still widely used to-day. It consists of the transference of pronator teres into the radial extensors of the wrist, flexor carpi ulnaris into the extensors of the third, fourth, and fifth digits, and flexor carpi radialis into extensor pollicis longus and extensor indicis. At about the same time as Robert Jones described his technique Stoffel (1918) in Germany was performing a similar operation, but used flexor carpi radialis as a transplant into the radial extensors, the tendon of flexor sublimis to the third digit for abductor pollicis longus and extensor pollicis brevis, and flexor carpi ulnaris for extensor pollicis longus and extensor communis digitorum. Starr (1922) was the first to suggest leaving one wrist flexor in its original place; he used flexor carpi radialis and palmaris longus to extend the digits, but did not disturb flexor carpi ulnaris. In five of his series of 43 cases of complete radial or posterior interosseous paralysis palmaris longus was absent, and rather than use both wrist flexors he split flexor carpi radialis into two and used one portion as transplant to the thumb and the other as transplant for the fingers. Although the earlier writers insisted on a transplant for the paralysed wrist extensors, Steindler (1940) does not advocate this procedure. He attaches flexor carpi radialis to extensor pollicis longus, palmaris longus to abductor pollicis longus, and flexor carpi ulnaris to extensor communis digitorum. Abbott (1944) reviewed the results of a series in which a slightly modified Robert Jones technique was used; pronator teres was attached to the radial extensors, flexor carpi radialis to all the extensors of the thumb and index, and flexor carpi ulnaris to the extensors of the other fingers. He found that in some cases there was powerful extension of the fingers, but in others this action was weak, and to remedy the defect a supplementary arthrodesis of the wrist was necessary. He concludes that "for those patients requiring hard usage of the wrist with complete extension of the fingers and thumb" a combined arthrodesis and tendon transplant is necessary. A summary of the various methods of tendon transplantation described above is given in *Table I*.

Table I.—Summary of Methods of Tendon Transplantation

Author	Date	Motor Tendon*	Recipient Tendon
Jones	1917	P.T. F.C.U. F.C.R.	E.C.R. E.C.D. to 3rd, 4th and 5th digits E.P.L. and E.I.
Stoffel	1918	F.C.R. Flexor sublimis to 3rd digit F.C.U.	E.C.R. A.P.L. and E.P.B. E.P.L. and E.C.D.
Starr	1922	(*a*) P.T. P.L. F.C.R. (*b*) P.T. F.C.R.	E.C.R. A.P.L., E.P.B., and E.P.L. E.I. and E.C.D. E.C.R. Extensors of all digits
Steindler	1940	F.C.R. P.L. F.C.U.	E.P.L. A.P.L. and E.P.B. E.C.D.
Abbott	1944	P.T. F.C.R. F.C.U.	E.C.R. Extensors of 1st and 2nd digits Extensors of 3rd, 4th, and 5th digits

* *Note*: The following abbreviations will be used for the muscles of the forearm in the tables :—

Pronator Teres	P.T.
Flexor Carpi Radialis	F.C.R.
Flexor Carpi Ulnaris	F.C.U.
Palmaris Longus	P.L.
Brachio-radialis	BR.
Extensor Carpi Radialis {Longus	E.C.R.L.
Extensor Carpi Radialis {Brevis	E.C.R.B.
Extensor Communis Digitorum	E.C.D.
Extensor Minimi Digiti	E. Min. Dig.
Abductor Pollicis Longus	A.P.L.
Extensor Pollicis Brevis	E.P.B.
Extensor Pollicis Longus	E.P.L.
Extensor Indicis	E.I.

Most of the authors stress the importance of careful technique and in particular the need for correct post-operative treatment. The persistent assertion in these papers that failures were due to defective post-operative care, strongly suggests that failures are by no means uncommon. The following analysis of tendon transplants for radial paralysis, performed at the Wingfield-Morris Hospital between 1940 and 1945, was undertaken with a view to determining the reasons why some tendon transplantations are less successful than others.

OPERATIVE AND POST-OPERATIVE TREATMENT

Although the operations were performed by different surgeons, the operative technique was the same in most respects, the principal variation being in the choice of tendons to be transplanted. The transplanted tendons were threaded through the paralysed tendons and sutured with black silk. In a few cases the tendons of the paralysed muscles were divided proximal to the site of suture to correct an extensor tendon contracture. The wrist and digits were held in extension while the suture was being performed and until a plaster cast had been applied and was firm. The wrist and metacarpophalangeal joints were maintained in extension, but the interphalangeal joints were allowed to flex to about 45°. After three weeks the plaster was removed and an anterior plaster slab applied; during the subsequent three weeks this splint was worn at night and for a variable period during the day, depending on the tightness of the transplant. Throughout this period the transplanted muscles were given faradic stimulation and voluntary exercise, and the patient was kept under close supervision.

* From the Department of Orthopædic Surgery, Oxford.

METHOD OF ASSESSING RESULTS

There is no generally accepted method of presenting the results of tendon transplantation, and reports of the quality of function after the operation are usually couched in terms such as good, fair, or poor, which have a variable connotation. A standard method of assessing the improvement in function after tendon transplantation is needed.

The functional deficiency in a radial paralysis is the inability to extend the wrist and digits, and the criterion of good recovery must be the return of these actions with minimal disturbance of other functions of the hand. It is not, however, necessary or even desirable to obtain full extension of the fingers when the wrist is dorsiflexed; such a position is unnatural and uncomfortable for most individuals. During the normal action of opening the clenched fist completely the wrist comes into the neutral or slightly flexed position, and the aim of the transplant must be to reproduce this action as closely as possible. It should allow full extension of the fingers with the wrist in the neutral or slightly flexed position and yet ensure active extension of the wrist when the fingers are flexed. Undesirable sequelæ of the operation would be defective flexion of the wrist or fingers.

The first important part of the examination is, therefore, an estimate of the range of extension of the fingers and thumb. Full active extension of the fingers against gravity is all that is required; incomplete extension of the fingers is a disability proportionate to the defective range. The essential range of movement of the thumb for general purposes is full extension of the digit when it lies midway between the plane of the palm and the plane of full palmar abduction. Any disability has again a direct relation to the defect in extension, but a detailed calculation of the disability is complex and it is sufficient to indicate whether extension of the thumb is full or not.

The second important assessment is the range and power of wrist extension; for good function a range of 20° is adequate, but the transplant must be strong enough to act against gravity and resistance, since on this depends the ability to lift heavy objects.

Defective flexion of the wrist can be assessed by measuring the range and power of this movement. The complete loss of the power of active flexion of the wrist is a disability of some magnitude, but if strong wrist flexion is retained, a limitation of the range of movement is of little consequence provided the neutral position can be attained. The strength of the digital flexors is not usually altered by the operation, but the range of flexion of the metacarpophalangeal joints may be limited and so contribute to a poor grip.

Examination for independent movements is principally a test of the neuromuscular control and adaptability of the patient. The fact that the patient can extend the thumb independently of the fingers does not imply independent action of the two transplants; nor does independent extension of a finger mean that one portion only of the transplanted muscle is acting. It is chiefly a matter of independent relaxation of the interossei and lumbricals.

The principal points to be considered in assessing the function of a tendon transplant can be summarized as follows:—

1. Range and power of extension of fingers and thumb.
2. Range and power of wrist extension.
3. Range and power of wrist flexion.
4. Range of flexion of fingers.

When an assessment has been made according to each of these criteria it is possible to specify function as a percentage of the optimum after transplantation. Each defect is allotted an arbitrary figure, and if the total for all defects is subtracted from 100 per cent a useful index of function is obtained. The following individual assessments are made:—

Defective extension of fingers: for each 10° short of full extension	10	per cent
Defective extension of thumb	10	,, ,,
Defective extension of wrist: inability to extend wrist to 20° against resistance	20	,, ,,
Defective flexion of wrist: inability to flex to neutral position	20	,, ,,
Defective flexion of fingers: slight	10	,, ,,
serious	20	,, ,,

It is clear that the value of any estimate obtained in this way depends primarily on how closely the figures correspond to reality. Supplementary methods of assessment are: first, determining what proportion of patients achieve full extension of the fingers and thumb and what proportion of patients can flex the wrist to the neutral position and extend it to 20° against resistance; secondly, by a series of photographs of transplants during these movements. It is not feasible to include photographs of every case, but a number of typical examples will be presented.

RESULTS

There were 22 cases of posterior interosseous paralysis and 35 of complete radial palsy. In four of the latter the wrist was arthrodesed; this group will be considered later. In the remaining 31 cases of complete radial paralysis, pronator teres was transplanted into the wrist extensors and 29 patients could extend the wrist to 20° against gravity and resistance, although in three the power of the transplant was significantly weak—$3\frac{1}{2}$*. In all respects other than restoration of wrist extension, the complete radial paralysis and the posterior interosseous paralysis present similar problems—namely, the return of extension of the digits while preserving flexion of the wrist and fingers. The results will be presented in two main groups: (*a*) Those in which both wrist flexors were transplanted; and (*b*) Those in which one wrist flexor only was used. These two groups, comprising 53 cases, will be considered first.

a. **Both Wrist Flexors Transplanted.**—In 24 cases both flexor carpi radialis and flexor carpi ulnaris were transplanted into the extensors of the digits. In *Cases* 1–5 (*Table II*) palmaris longus was absent and the results were deplorable. Not one

* The method of grading muscle power is that recommended by the Medical Research Council. 0 = complete paralysis; 1 = flicker of voluntary action; 2 = voluntary action sufficient to move the joint; 3 = action against gravity; 4 = action against gravity and resistance; 5 = full power.

of the patients could actively flex the wrist to the neutral position nor extend the thumb or fingers completely. The defective range of extension of the fingers varied between 30° and 60°. Yet, when the wrist was passively maintained in a neutral position, full active extension of the fingers was possible. There was thus no doubt that the transplant had been performed under adequate tension and that it would function satisfactorily if the wrist could be maintained in the position it normally occupies during the movement. The results, expressed as a percentage, vary between 10 per cent and 40 per cent, with an average of 26 per cent.

In *Cases* 6–24 (*Table III*), palmaris longus was present and was left in position. Although the results are better than those in *Table II*, many are not satisfactory. In 14 patients out of 19 the defective range of extension of the metacarpophalangeal joints was more than 10°, and in 5 patients it was more than 40°. In 15 cases the range of extension of the thumb was defective. Eight patients could not flex the wrist to the neutral position. The percentage grading of function varies between 20 per cent and 90 per cent, with an average of 57 per cent. The combined results in these 24 cases in which both wrist flexors were transplanted average 50 per cent, with a standard error of ± 5·0.

b. **Only One Wrist Flexor Transplanted.**—In the remaining 29 patients flexor carpi radialis was left in situ (*Table IV*). Flexor carpi ulnaris was used as motor tendon to the extensors of the fingers in all cases except one—where extensor carpi radialis longus was employed. The transplant to the thumb extensor varied: in 5 cases brachioradialis was used and in 2 an active wrist extensor: in 12 patients in whom palmaris longus was present this tendon was used for extension of the thumb; but 9 patients had no palmaris longus, and for them flexor carpi ulnaris was transplanted to the thumb extensors as well as those of the fingers.

Twenty-seven out of 29 patients could extend the fingers to the neutral position; 18 patients could extend the thumb completely; and all could flex the wrist strongly to the neutral position. In one patient flexion of the index was limited. The functional result expressed as a percentage varied between 70 per cent and 100 per cent, with an average of 91 per cent and a standard error of ± 1·4. The difference between this figure and the result of 50 per cent for the previous group of cases is almost eight times the standard error and is therefore unlikely to be due to chance.

The comparison of the results in the two groups is shown in another way in *Table V*. In only 21 per cent of those cases in which both wrist flexors were transplanted was almost full extension of the fingers possible, compared with 97 per cent of those in which only one wrist flexor was used. Of the former group 46 per cent could flex the wrist to the neutral position compared with 100 per cent of the latter. Although there was not such a marked contrast in the range of extension of the thumb in the two groups of cases, the difference between 16·5 per cent in the former and 69 per cent in the latter is far more than twice the standard error and therefore must be regarded as of considerable significance.

Table II.—Summary of Results when both Wrist Flexors were Transplanted, but Palmaris Longus was Absent

No.	File No.	Transplant		Remaining Wrist Stabilizer	Results								Comment
					Extension of Wrist		Flexion of Wrist		Extension of Thumb		Extension of Fingers		Function Expressed as Percentage	
		Motor Tendon	Recipient Digit		Range in Degrees	Power	Range in Degrees	Power	Range in Degrees	Power	Range in Degrees*	Power		
1	W.80	F.C.R. F.C.U.	1–2 2–5	None	–	–	Nil	Nil	Limited	4	60	4½	10	Improved by fixation of wrist
2	M.44	P.T. F.C.R. F.C.U.	E.C.R. 1–2 3–5	None	30–60	4	Nil	Nil	Limited	3½	50	3½	20	Needs fixation
3	M.42	P.T. F.C.R. F.C.U.	E.C.R. 1–2 3–5	None	10–40	4	Nil	Nil	Limited	4	40	4	30	
4	G.15	P.T. F.C.R. F.C.U.	E.C.R. 1–2 3–5	None	40–60	3½	Nil	Nil	Limited	2½	40	4	30	Cannot let go
5	M.14	P.T. F.C.R. F.C.U.	E.C.R. 1 2–5	None	30–60	5	Nil	Nil	Limited	5	30	5	40	Desires fixation

* The figures indicate the number of degrees short of full extension.

Table III.—SUMMARY OF RESULTS WHEN BOTH WRIST FLEXORS WERE TRANSPLANTED, AND PALMARIS LONGUS WAS PRESENT

No.	File No.	Transplant		Remaining Wrist Stabilizer	Results									
					Extension of Wrist		Flexion of Wrist		Extension of Thumb		Extension of Fingers		Function Expressed as Percentage	Comment
		Motor Tendon	Recipient Digit		Range in Degrees	Power	Range in Degrees	Power	Range in Degrees	Power	Range in Degrees*	Power		
6	L.36	P.T. F.C.R. F.C.U.	E.C.R. 1 2–5	P.L.	Nil	Nil	30	4	Limited	3	40	3	20	P.T. transplant slipped—flexion of fingers limited. Arthrodesis of wrist
7	H.57	P.T. F.C.R. F.C.U.	E.C.R. 1–2 2–5	P.L.	40	4½	Nil	Nil	Limited	4½	45	4½	25	
8	N.9	P.T. F.C.R. F.C.U.	E.C.R. 1 2–5	P.L.	40	4½	Nil	Nil	Limited	4½	45	2½	25	
9	H.1	P.T. F.C.R. F.C.U.	E.C.R. 1 1–5	P.L.	30	4	Nil	Nil	Limited	4	30	3½	40	
10	M.29	P.T. F.C.R. F.C.U.	E.C.R. 1 2–5	P.L.	60	4½	Neutral	3	Limited	5	35	4½	45	Desires fixation of wrist
11	G.49	F.C.R. F.C.U.	1 2–5	P.L.			Nil	Nil	Full	4½	30	4	50	
12	H.82	F.C.R. F.C.U.	1 2–5	P.L.			25	4	Limited	5	40	5	50	
13	V.1	P.T. F.C.R. F.C.U.	E.C.R. 1–2 3–5	P.L.	40	4½	Nil	Nil	Limited	4½	20	4½	50	
14	P.54	P.T. F.C.R. F.C.U.	E.C.R. 1–2 2–5	P.L.	25	4	20	4½	Limited	3	40	4	50	
15	E.6	P.T. F.C.R. F.C.U.	E.C.R. 1 2–5	P.L.	20	4	Nil	Nil	Limited	4	15	4	55	
16	H.28	F.C.R. F.C.U.	1 2–5	P.L.			Nil	Nil	Limited	3	10	4½	60	
17	Q.2	P.T. F.C.R. F.C.U.	E.C.R. 1–2 2–5	P.L.	40	4	20	4	Limited	3½	30	3½	60	Thumb in way. Arthrodesis of wrist
18	F.44	P.T. F.C.R. F.C.U.	E.C.R. 1 2–5	P.L.	40	4½	—10	3	Limited	4½	15	4½	65	
19	M.46	P.T. F.C.R. F.C.U.	E.C.R. 1–2 2–5	P.L.	30	4½	10	3½	Limited	3½	25	4½	65	
20	L.38	P.T. F.C.R. F.C.U.	E.C.R. 1–2 2–5	P.L.	35	4	20	3½	Limited	4	15	3½	75	
21	W.77	P.T. F.C.R. F.C.U.	E.C.R. 1 2–5	P.L.	25	4	5	4	Limited	4½	10	4	80	
22	E.21	P.T. F.C.R. F.C.U.	E.C.R. 1 2–5	P.L.	30	4	20	4	Full	4	10	4½	90	
23	B.112	P.T. F.C.R. F.C.U.	E.C.R. 1 2–5	P.L.	35	4½	20	4	Full	4½	10	4½	90	
24	H.88	P.T. F.C.R. F.C.U.	E.C.R. 1 2–5	P.L.	25	4½	15	3	Full	4½	10	4½	90	

* The figures indicate the number of degrees short of full extension.

* The figures indicate the number of degrees short of full extension.

Table IV.—Summary of Results when One Wrist Flexor was Transplanted

No.	File No.	Transplant		Remaining Wrist Stabilizer	Results									
					Extension of Wrist		Flexion of Wrist		Extension of Thumb		Extension of Fingers		Function Expressed as Percentage	Comment
		Motor Tendon	Recipient Digit		Range in Degrees	Power	Range in Degrees	Power	Range in Degrees	Power	Range in Degrees*	Power		
25	H.24	E.C.R.B. F.C.U.	1–2 3–5	F.C.R.			Neutral	4	Limited	5	Full	5	70	Index tight
26	R.49	P.T. P.L. F.C.U.	E.C.R. 1–2 2–5	F.C.R.	20	3½	20	4	Full	3½	10	3½	70	Wrist extension weak
27	H.124	BR. F.C.U.	1 2–5	F.C.R.			20	4	Full	4½	Full	4½	80	Wrist in radial deviation
28	G.80	P.T. P.L. F.C.U.	E.C.R. 1 2–5	F.C.R.	25	4	50	4	Limited	4½	Full	4	80	Hand in slight ulnar deviation
29	R.66	P.T. P.L. F.C.U.	E.C.R. 1 2–5	F.C.R.	20	3½	25	4½	Full	3½	Full	4	80	Wrist extension weak
30	H.116	BR. F.C.U.	1 2–5	F.C.R.			20	4½	Full	3	Full	4½	90	
31	B.172	BR. F.C.U.	1 2–5	F.C.R.			10	4	Limited	3½	Full	3½	90	
32	C.8	BR. F.C.U.	1 2–5	F.C.R.			45	4	Limited	3	Full	5	90	
33	C.44	E.C.R.L. F.C.U.	1–2 3–5	F.C.R.			45	4	Limited	4	Full	4	90	
34	D.11	F.C.U. E.C.R.L.	1 2–5	F.C.R.			Neutral	5	Full	4½	Full	4½	90	Wrist flexion prevented by tight extensors
35	S.132	P.L. F.C.U.	1 2–5	F.C.R.			Neutral	3½	Full	4	Full	4	90	Wrist flexion limited 8 weeks after operation
36	R.31	P.L. F.C.U.	1 2–5	F.C.R.			Full	4½	Limited	3	Full	3½	90	
37	H.7	F.C.U.	1–5	F.C.R.			15	4½	Full	5	2nd Full 3, 4, 5, 15°	5	90	
38	F.56	F.C.U.	1–5	F.C.R.			20	5	Limited	4	Full	4	90	
39	E.22	P.T. P.L. F.C.U.	E.C.R. 1 2–5	F.C.R.	40	4½	40	4	Limited	4½	Full	4½	90	
40	B.135	P.T. P.L. F.C.U.	E.C.R. 1 2–5	F.C.R.	30	4	10	4	Full	4	Full	4	90	
41	S.51	P.T. F.C.U.	E.C.R. 1–5	F.C.R.	40	4	80	5	Limited	4½	Full	4½	90	
42	C.84	P.T. F.C.U.	E.C.R. 1–5	F.C.R.	40	4	25	4½	Limited	3½	Full	4½	90	
43	R.62	P.T. F.C.U.	E.C.R. 1–5	F.C.R.	25	4	20	4	Limited	3½	Full	4	90	
44	B.162	BR. F.C.U.	1 2–5	F.C.R.			25	4½	Full	5	Full	5	100	
45	L.35	P.L. F.C.U.	1 2–5	F.C.R.			Full	3½	Full	4	Full	4½	100	
46	L.51	P.L. F.C.U.	1 2–5	F.C.R.			35	4	Full	3½	Full	4½	100	
47	C.131	P.L. F.C.U.	1 2–5	F.C.R.			15	4	Full	4	Full	4	100	
48	W.45	F.C.U.	1–5	F.C.R.			10	4½	Full	4	Full	5	100	
49	R.41	F.C.U.	1–5	F.C.R.			20	4½	Full	4½	Full	4½	100	
50	G.76	F.C.U.	1–5	F.C.R.			40	4½	Full	4	Full	4	100	
51	J.56	P.T. P.L. F.C.U.	E.C.R. 1 2–5	F.C.R.	25	4	10	4½	Full	4	Full	4	100	
52	B.138	P.T. P.L. F.C.U.	E.C.R. 1 2–5	F.C.R.	20	4½	20	4	Full	4½	Full	4½	100	Playing piano
53	Mc.22	P.T. F.C.U.	E.C.R. 1–5	F.C.R.	90	4	30	4	Full	4	Full	4	100	

* The figures indicate the number of degrees short of full extension.

Table V.—COMPARISON OF RESULTS IN *Tables II–IV*

	Extension of M.P. Joints to 10°		Flexion of Wrist to Neutral		Flexion of Wrist to 20°		Full Extension of Thumb		Total No. of Cases
	No. of Cases	Percentage	No. of Cases	Percentage	No. of Cases	Percentage	No. of Cases	Percentage	
a. Both wrist flexors transplanted	5	21	11	46	7	29	4	16·5	24
b. One wrist flexor transplanted	28	97	29	100	20	69	18	62	29

Table VI.—RESULTS AFTER FIXATION OF WRIST

No.	File No.	Transplant		Remaining Wrist Stabilizer	Results					
					Extension of Thumb		Extension of Fingers		Function Expressed as Percentage	Comment
		Motor Tendon	Recipient Digit		Range in Degrees	Power	Range in Degrees*	Power		
54	C.25	P.L. F.C.U.	1 2–5	Arthrodesis	Full	3½	30	3½	70	
55	E.30	P.L. F.C.R.	1 2–5	Tenodesis failed Arthrodesis	Limited	3½	20	4	70	Tenodesis a failure—wrist arthrodesed
56	H.58	P.L. F.C.R.	1 2–5	Arthrodesis	Nil	Nil	Nil	Nil	Nil	Post-operative hæmatoma and adhesions. The transplanted muscles were not strong.
57	B.63a	F.C.R. F.C.U.	1 2–5	Arthrodesis	Full	4	Full	4	100	

* The figures indicate the number of degrees short of full extension.

The adverse effect of transplanting both wrist flexors is illustrated in the series of photographs. In *Figs.* 413–416 both wrist flexors were used and the fingers. In *Figs.* 421–427 are shown those cases in which flexor carpi radialis was left in situ. It is at once evident that extension of the fingers is

FIG. 413.—*Case* 2. Palmaris longus absent; both wrist flexors transplanted. Attempted extension of fingers and thumb. Note the marked dorsiflexion of the wrist and incomplete extension of the metacarpo-phalangeal joints.

FIG. 414.—*Case* 2. Extension of fingers improved if dorsiflexion of wrist is prevented.

palmaris longus was absent: the dorsiflexion of the wrist which accompanies the attempt to extend the fingers is well illustrated, and passively maintaining the wrist in the neutral position allows active extension of the fingers to take place. In *Figs.* 417–420 palmaris longus was present and in the first picture it appears to stabilize the wrist fairly well and permit satisfactory extension of the fingers. In *Figs.* 418 and 419 it is seen to be less effective and does not prevent hyperextension of the wrist; and *Fig.* 420 shows that adequate support for the wrist to prevent this excessive dorsiflexion permits full extension of more complete in this series and strong wrist flexion is preserved (*Figs.* 422 and 426). *Fig.* 427 shows the effect of transplanting flexor carpi ulnaris into the fingers and thumb in a patient who had no palmaris longus: satisfactory extension of all digits is obtained and the wrist remains in the natural position during the movement.

c. **Fixation of Wrist.**—In 4 patients the wrist was fixed (*Table VI*)—in 3 by operation, and in 1 by trauma. One was a complete failure in which weak muscles had been transplanted and a post-operative hæmatoma led to adhesions.

FIG. 415.—*Case* 1. Palmaris longus absent; both wrist flexors transplanted. Attempted extension of fingers and thumb. Note the marked dorsiflexion of the wrist and incomplete extension of the metacarpo-phalangeal joints.

FIG. 416.—*Case* 1. Extension of fingers improved if dorsiflexion of wrist is prevented.

FIG. 417.—*Case* 22. Palmaris longus present; both wrist flexors transplanted. Almost full extension of fingers.

FIG. 418.—*Case* 7. Palmaris longus present; both wrist flexors transplanted. Incomplete extension of fingers and marked dorsiflexion of wrist.

FIG. 419.—*Case* 12. Palmaris longus present; both wrist flexors transplanted. Incomplete extension of fingers and marked dorsiflexion of wrist.

FIG. 420.—*Case* 12. Almost complete extension of fingers if dorsiflexion of wrist is prevented.

In two patients extension of the fingers was limited by 20° and 30°, but in one a full range was possible. Two of the three had full extension of the thumb.

DISCUSSION

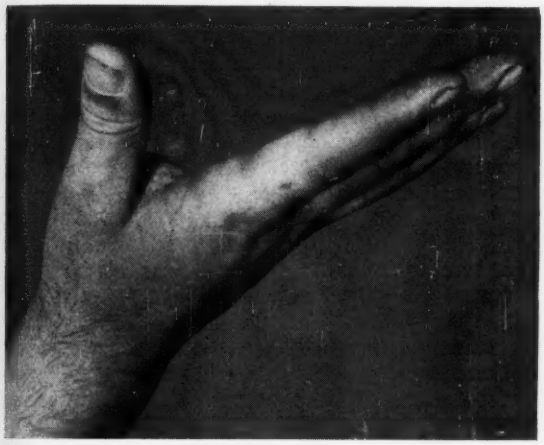
There are two important questions to be considered in the choice of tendon for transplantation: (1) How closely can the transplanted muscle simulate the action of the paralysed one? (2) Will the transference of the tendon cause undesirable sequelæ?

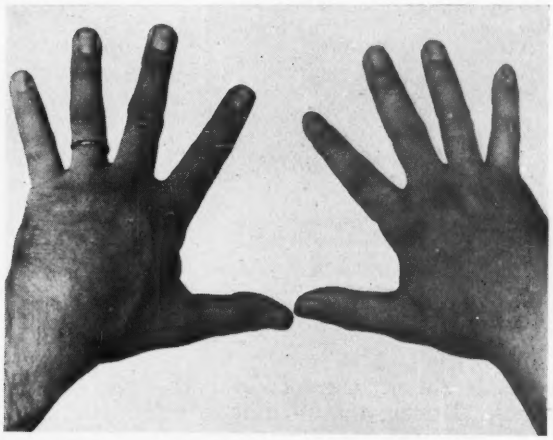
1. The ability of a muscle to take over the action of another depends on its strength, its range of contraction, and the possibility of re-education.

a. The strength of a normal muscle depends on the number and obliquity of the muscle-fibres. The greater their number and the smaller the angle they make with the tendon, the greater will be the power.

FIG. 421.—*Case* 44. Palmaris longus present; one wrist flexor transplanted. Complete extension of fingers and thumb.

FIG. 422.—*Case* 44. Strong flexion of wrist against resistance, showing flexor carpi radialis and palmaris longus in situ.





FIGS. 423, 424.—*Case* 51. Palmaris longus present; one wrist flexor transplanted. Complete extension of fingers and almost complete extension of thumb.

FIG. 425.—*Case* 46. Palmaris longus present; one wrist flexor transplanted. Complete extension of fingers and thumb.

FIG. 426.—*Case* 46. Strong wrist flexion against resistance.

b. The range of action depends on the length and direction of the muscle-fibres and the direction of pull of the transplanted tendon. The shorter the fibre and the greater the angle it makes with the tendon, the smaller will be the range of movement of the tendon. The greater the angle at which the transplant joins the paralysed tendon, the smaller the range of action. Conversely, the longer the fibres, the more longitudinally they run, and the more closely the motor and paralysed tendons run parallel, the greater will be the range of action.

c. In a review dealing with nerve and muscle transposition, Sperry (1944) quotes a number of reports of failures of transplantation when antagonistic muscles were used. There is, however, ample evidence that the wrist flexors can take over the

action of muscles to which they are synergists—the extensors of the fingers—but we do not know with what facility or how completely the necessary re-adaptation may be attained. However, in two of the cases reported above, flexor carpi ulnaris had become so thoroughly re-educated that six weeks after transplantation the electromyographic recording from this muscle showed no action potentials during active flexion of the wrist. Since such a complete change of action is possible it seems unlikely that the partial readjustment of a synergist will present any difficulties.

2. The transference of pronator teres into the wrist extensors may cause a little limitation of supination of the forearm, but the muscle still maintains a powerful pronating action. It is a different matter with the wrist flexors. If palmaris

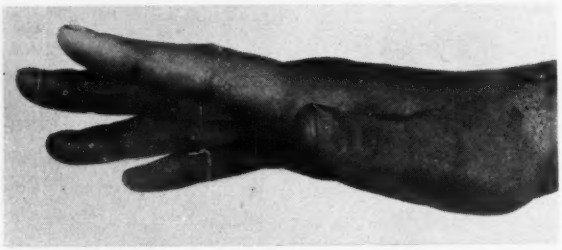


FIG. 427.—*Case* 48. Palmaris longus absent; one wrist flexor transplanted into all digits. Full extension of fingers and thumb.

longus is absent, the loss of both wrist flexors is serious. During the attempt to extend the fingers the transplanted tendons, passing behind the wrist and being unopposed, are bound to extend it. A transplanted muscle, like any other, has only a limited range of action and on account of the obliquity of its course its range is diminished; if the length of contraction of the fibres is used up in extending the wrist, there will be little left to extend the digits. This is the explanation of the defective range of extension in several cases in which both flexor carpi radialis and flexor carpi ulnaris were used as transplants.

It is sometimes contended that the long flexors of the fingers can act as flexors of the wrist and that with re-education the control of the wrist should be satisfactory. It is certainly true that the finger flexors may flex the wrist at the same time as they flex the fingers, but it is equally clear that they will be unable to perform this function at the same time as the fingers are extended, since the finger flexors are reciprocally relaxed during this action. The absence of the wrist flexors leads to excessive dorsiflexion of the wrist when the patient attempts to extend the fingers; when such a transplant is performed it is just possible to obtain full extension of the metacarpo-phalangeal joints in spite of the unnatural position of the wrist—but only by performing the operation under such tension that flexion of the wrist and fingers is grossly limited.

Clearly, then, it is desirable to retain a wrist flexor. If palmaris longus is absent, only flexor carpi ulnaris can be spared for transplantation, unless the wrist is controlled in some other way, e.g., by arthrodesis. In the case of a posterior interosseous paralysis there are sufficient tendons available without removing a second wrist flexor, but in a complete radial paralysis the only alternative to arthrodesis of the wrist is to use flexor carpi ulnaris as transplant to all the digits. This has been shown to be satisfactory.

If palmaris longus is present the choice lies between this tendon and flexor carpi radialis as transplant. It has been demonstrated that, even when palmaris longus was left behind, the control of the wrist was not always effective and full extension of the fingers was not invariable. Flexor carpi radialis always controlled the wrist effectively enough to permit complete or almost complete extension of the metacarpo-phalangeal joints, and, moreover, preserved strong wrist flexion to the neutral position or further.

CONCLUSION

In tendon transplantation for radial paralysis it is important to preserve an active wrist flexor to stabilize the wrist. The neglect of this precaution often leads to incomplete extension of the fingers and unnecessarily weakens or abolishes the power of wrist flexion. If flexor carpi radialis is left in situ these sequelæ seldom arise.

SUMMARY

1. A standard method of assessing the results of tendon transplantation for radial paralysis is suggested.

2. The results of tendon transplantation are described in 57 cases of radial paralysis.

3. Better results were obtained by transplanting only one wrist flexor, flexor carpi ulnaris, and leaving flexor carpi radialis in situ.

My thanks are due to Professor H. J. Seddon for his valuable advice in the preparation of this manuscript.

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A CASE OF SEVERE ELECTRIC BURNS WITH AN UNUSUAL SEQUENCE OF COMPLICATIONS

By JULIAN SMITH, Lt.-Col., A.A.M.C., and B. K. RANK, Lt.-Col., A.A.M.C.

A complete record of the clinical details and investigations concerning the case of one, L.A.C. T. H. Van A., aged 18, of the R.A.A.F., would be tiresome if presented *in extenso*. The following extract, prolonged as it may appear, contains its salient features only, and we will not repeat in the text those details which are graphically shown in *Fig.* 428.

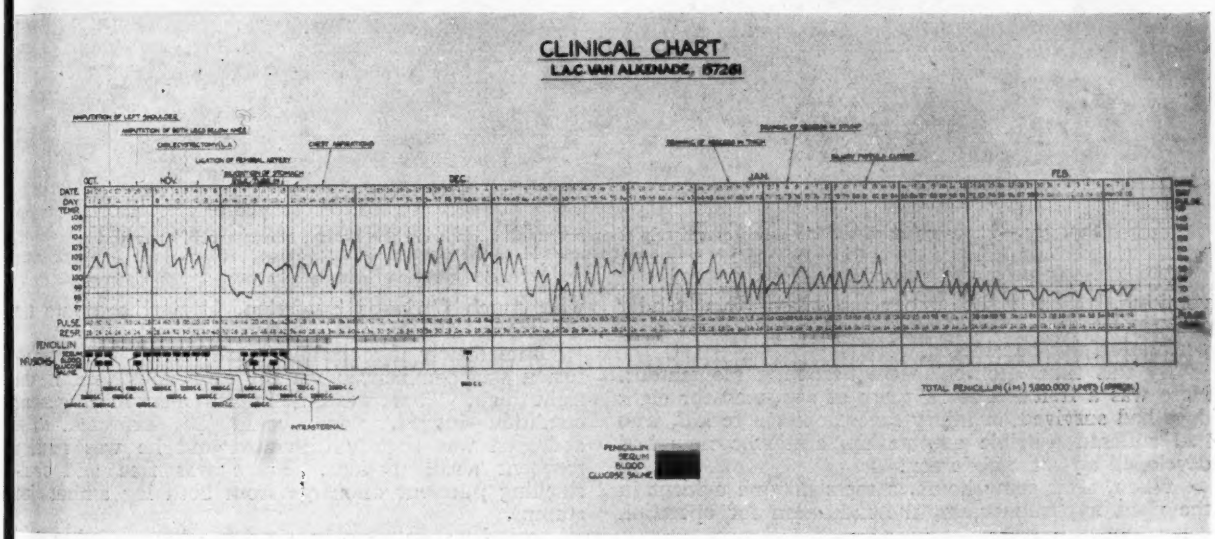


Fig. 428.—Reproduction of clinical chart—temperature, pulse respiration, record of penicillin therapy, and transfusions.

CASE REPORT

Oct. 23, 1944: At 17·00 hr. L.A.C. Van A. was one of two survivors when a mobile crane on which he was working fouled high-tension electric wires. In the flash, he was thrown some distance, and it was immediately obvious that he had sustained burns to both lower limbs and the left arm.

He was taken to No. 2 R.A.A.F. Hospital. On admission, the clinical notes stated: "pulse 120, moaning and restless. Right foot cold and indurated, with burns extending to the knee. Left foot indurated with exposed and charred metatarsals. Left arm white and indurated up to the shoulder—several phalanges of the hand exposed and charred."

Five hours after the accident, infusion of serum was commenced, and under general anæsthesia the burnt areas were cleansed and dressed.

2nd day: He was transferred to No. 2 Plastic Surgery Unit (on the advice of W/Cdr. Searby), as it was considered that a radical excision of the burnt areas of skin with replacement might have been feasible.

At this time—17 hours after the injury—the pulse was 140, weak but regular. Blood-pressure 110 systolic. Muttering delirium. Hæmoglobin 105 per cent, hæmatocrit 51 per cent, plasma-protein 7·2 per cent.

On inspection of the burnt areas, it was apparent that both feet were lifeless, the soft tissues being either charred or coagulated. On the right leg, second-degree burns extended above this level to the knee. The left upper extremity was completely mummified, except for an area of skin over the deltoid region.

His general condition continued to be grave. Blood examination revealed 3,500,000 red cells per c.mm., hæmoglobin 69 per cent, hæmatocrit 30 per cent, plasma-protein 6·5 per cent. He had had 5 litres of serum since the injury. Blood transfusion was now commenced.

The lad, by this time, had survived for two days. All efforts had so far been directed to providing the usual restorative measures which abundant laboratory investigations indicated were expedient. At this juncture we felt that, despite adequate resuscitation, recovery could not be expected if the large areas of dead tissue in the extremities were allowed to remain.

First Operation.—Under nitrous-oxide anæsthesia, the left arm was removed through the shoulder-joint. Nerve-trunks were injected with novocain. The available deltoid flap was used to cover the defect and a tube was left in situ for penicillin instillation. The amputated limb is illustrated in *Fig.* 429.

At operation, deep coagulation of the tissues was demonstrated up to the level of amputation. There was extreme œdema and reaction about the shoulder.

3rd day: By this time he had been given 3 litres of blood. Red blood-cells 4,200,000, hæmoglobin 80 per cent, hæmatocrit 39 per cent, plasma-protein 6·5 per cent. Glucose-saline infusion was therefore substituted.

6th day: Despite the fact that he had withstood the first operation as well as could be expected, his general condition was deteriorating. Both legs were offensive.

Second Operation.—Under cyclopropane anæsthesia, the left leg was amputated through the middle third at the level of the skin loss, the flaps being loosely sutured over the bone ends and drain tubes inserted. The right leg was amputated at a higher level, and in order to conserve adequate length of limb for a below-knee prosthesis, the stump included areas of burnt, but not dead, skin. Flaps were also loosely sutured and drains and tubes for penicillin inserted.

At this stage, our slight feelings of optimism engendered by the return, for the first time, of the lad's mental

faculties, were rudely dispersed by a severe epistaxis and the onset of abdominal pain.

8th day: General condition poor. Complaining of severe abdominal pain, colicky in nature, felt in the right lower abdomen. It was accompanied by diarrhœa and vomiting. The right upper abdomen was tender and rigid. The possibility of acute peptic ulcer with perforation was considered.

By this time decisions were becoming momentous. Here was a frail lad of 18 years of age, who for eight days had survived an injury severe enough to kill, who had suffered a triple amputation, and who now had developed an acute abdomen.

When, after some hours, a mass became evident in the right hypochondrium, the indication for operation was abundantly clear.

THIRD OPERATION.—Under local anæsthesia, a right upper paramedian incision was made, and the abdominal cavity opened. There was a fair amount of fluid and exudate surrounding a large distended gall-bladder, the fundus of which included multiple gangrenous areas. Dark, thick bile was aspirated and a partial cholecystectomy performed. A tube was sutured into the remaining part of the viscus. A second tube was lead into Morison's pouch via a stab wound in the flank. The abdomen was closed in layers.

The specimen is reproduced in *Fig.* 430.

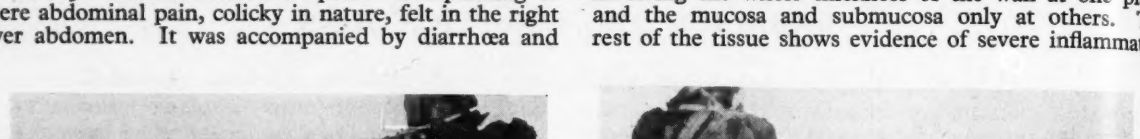


FIG. 429.—Photographs of the left arm immediately after amputation, showing the typical admixture of char and coagulation of the skin and tissues. Note the exposed bone.

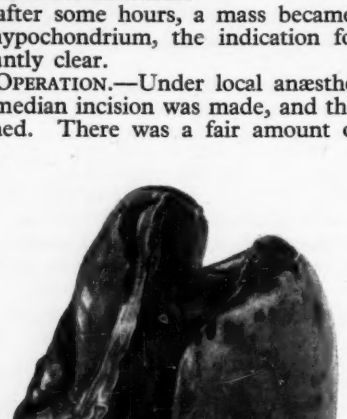


FIG. 430.—Coloured reproduction of the gall-bladder as it appeared after removal. Note three separate infarcts, the large one involving the full thickness of the gall-bladder wall.

On the microscopical sections of the gall-bladder the pathologist reported "Coagulative necrosis has occurred, involving the whole thickness of the wall at one place, and the mucosa and submucosa only at others. The rest of the tissue shows evidence of severe inflammation with much fibrinous exudation, hæmorrhage into the tissues, and accumulation of polymorphs."

Immediately after the abdominal section, blood transfusion was commenced via the saphenous vein in the right thigh. There was little improvement in his general condition—indeed, none could be expected. The abdomen was slightly distended and he was passing frequent small motions. There was free and foul-smelling purulent discharge from both leg amputation stumps.

12th day: At 08·00 hr. six days after the lower limb amputations, there was a vigorous secondary hæmorrhage from the stump of the left leg, controllable only by tourniquet. This complication was not unexpected.

FOURTH OPERATION.—Under cyclopropane anæsthesia, the amputation stump was opened, clot removed, but no bleeding point could be found. The femoral artery was ligated in Hunter's canal, and blood transfusion recommenced in the right arm.

14th day: Free drainage of bile from the abdominal wound continued. All amputation stumps were discharging freely and culture showed—"Leg stump: *Bact. coli*, *Ps. pyocyanea*, *Proteus morganii*. L. arm: *Bact. coli*, *Ps. pyocyanea*, *Proteus vulgaris*, hæmolytic *Staph. aureus*, green zone streptococci".

The leg stumps were suspended in Thomas splints and continuous irrigation with Eusol arranged to alternate with hypertonic saline.

17th day: General condition very poor. The diarrhœa had ceased, but was replaced by copious vomiting. A stomach tube was inserted. Intravenous serum was again instituted by way of a vein on the dorsum of the right hand.

Continuation of intravenous therapy was now a problem because he was suffering gross fluid loss. The right arm was very swollen and œdematous. All available arm veins had been used, as had both saphenous veins in the upper thighs. Sternal puncture was therefore performed and glucose-saline given. Aspiration of the stomach continued to yield large quantities of fluid. The sternal drip continued to run satisfactorily after four days.

21st day: Coughing up blood-stained sputum. Radiography revealed bilateral pneumonia which had been suggested by clinical signs. Happily, at this time, we were able to dispense with the gastric suction.

Dyspnœa, however, became more marked, and both pleural cavities were aspirated—the right side yielding 50 oz. of blood-stained fluid, the left side 18 oz. Chest aspiration was continued twice daily. The fluid remained straw-coloured and contained blood-cells and leucocytes. Cultures of repeated specimens revealed no growth.

24th day: For the first time, a decided general improvement could be noticed. The vomiting had ceased. The abdomen was satisfactory, although the biliary fistula persisted with copious discharge. Chest aspirations yielded similar fluid, but in decreasing amounts. There was a heavy Gram-negative infection of all wounds. Intravenous therapy had been suspended.

During the next three weeks the general condition slowly improved, although the temperature chart remained ugly. The lad had innumerable routine examinations and special investigations referable to all systems, in an effort to determine the cause of the persistent high fever. He was seen by many specialists and consultants. Blood-cultures repeatedly showed "no growth". He was given additional blood transfusions and no end of medicinal and dietetic therapy.

66th day: A diffuse flat indurated mass in the left trochanteric region was noted in a routine examination.

FIFTH OPERATION.—Under nitrous-oxide and oxygen anæsthesia, the swelling was needled and a small amount of very thick pus obtained. Through a lateral thigh incision, deep to the fascia femoris, a large loculated collection of pus about the femur was displayed, and 1½ pints evacuated. Culture yielded a pure growth of *B. coli*.

73rd day: Low-grade cellulitis with deep induration was noticed in the left leg stump.

SIXTH OPERATION.—Under gas anæsthesia the stump was incised and a deep collection of pus was evacuated.

After these two abscesses had been drained the temperature gradually fell to normal.

Three months after the accident he was sitting up in a chair. His weight was 6 st. 10 lb. The biliary fistula finally closed. Radiography of chest was clear. There was a small sinus in the shoulder stump, but the leg stumps had healed after a small sequestrum had been removed from the right leg stump.

He was measured for his artificial legs, and transferred to No. 2 R.A.A.F. Con. Depot, Warburton. During his month's convalescence he had periods of marked depression, and a further exacerbation of low-grade infection with abscess formation in the shoulder wound. He returned to hospital and was fitted with his artificial legs, which he managed very well right from the start.

Finally, he was discharged (*Fig.* 431) in very good general condition, and maintaining a philosophic attitude with regard to the loss of his limbs which was admirable. He regards himself as happily placed in the Repair Department of the State Electricity Commission, where he is a switchboard operator.

DISCUSSION

1. *Triple amputation of limbs for severe electric burn*: The value of early local excision for electric burns, in view of their depth and localized nature, is well recognized and widely practised. Extension of this principle to the degree of amputating three limbs for an extreme degree of electro-coagulation cannot be common. The course followed in this case was dictated by surgical necessity.

2. *The problem of fluid infusion*: In this case, where we were denied the use of both legs and an upper extremity, the maintenance of adequate infusions was indeed a problem. This lad had an almost continuous series of blood, serum, and glucose-saline infusions over a period of 21 days. When, at his most critical stage, all the usual channels in the remaining limb parts had been exhausted, the use of a continuous intrasternal drip for 4 days, during the period of acute dilatation of the stomach, was able to maintain his fluid balance. This *per se* was a life-saving measure. We would therefore suggest that in any case where infusions are likely to be multiple or protracted it is wise to select veins

FIG. 431.—Patient on discharge from hospital, 7 months after the injury.

systematically. Main venous trunks should not be used and occluded early in the programme. When confronted with this problem, the sternal route—at least for glucose-saline—is most valuable.

3. *The gall-bladder complication*: That such a lesion of the gall-bladder can occur as a complication of burns may in itself be worthy of record. After searching the literature we have been unable to find a case of such a complication following burns of this type. The discrete appearance of the three gangrenous patches (*see Fig.* 430) would suggest an embolic basis for the condition. It would seem that such patches are of similar origin and nature to the so-called Curling's ulcer of the duodenum which is encountered more commonly as a complication of burns. The common basic blood-supply of the gall-bladder and duodenum may be significant.

4. *The aborted empyema*: The transient nature of the severe pulmonary complication and the fact that the pleural exudate remained sterile throughout, must be attributed to penicillin.

5. *The silent abscess*: This case occurred in the early days of penicillin therapy when its use was

perforce somewhat restricted. Parenteral penicillin was not commenced until the upper limb amputation was performed, but it was thereafter continued 3-hourly (with one short break) for 5 weeks.

Considering the large number of injections, the low ebb of the lad's vitality, and the multiplicity of his infected wounds, we were not unduly concerned when he developed an abscess at one of the injection sites, although it must be assumed that the infection was introduced. This complication, censurable under ordinary circumstances, was a triviality compared with the big issue at stake. The loculation and inspissated nature of pus in this abscess pointed to its long duration. It was certainly the cause of the persistent pyrexia during the latter part of this stormy passage.

6. *The result*: This lad's survival was not a tribute to any one factor. Thirty-four litres of saline, serum, and blood infusions; more than 5,000,000 units of penicillin; the highest standard of excellence in nursing; expert anæsthesia; some competent surgery, perhaps; and most certainly the fighting spirit of the lad himself—all played their part and contributed to his recovery. However, none of these factors could become contributors to the happy outcome, without the binding influence of surgical pertinacity, the will to 'go for' a result, and to shun any attitude of fatalism.

Faced now with an ardent young man, interested in all that a lad of eighteen should be, with his eyesight, his right hand, and his self-respect, we are obliged to forget the chides of many—"Thou shalt not strive officiously to keep alive".

SUMMARY

1. The salient features of a case of severe electrocoagulation burns treated by multiple amputation and followed by uncommon complications, with ultimate survival, is given.

2. Various features of this case are discussed *seriatim*: the triple amputation, the problem of fluid infusion, the gall-bladder complication, the aborted empyema, the silent abscess, and the ultimate result.

Though fully implied in the results obtained, we wish to acknowledge the order of work done by all the staff of the No. 2 Facio-maxillary and Plastic Surgery Unit in the difficult surgical management and the meticulous nursing of the case.

The illustrations are the work of Flight Officer Alldritt, artist, and Cpl. Hale, photographer, of this unit.

The permission of Director-General, Medical Services, Royal Australian Air Force, Air Vice-Marshal T. E. V. Hurley, to publish this report is acknowledged.

THREE CASES OF FRACTURE-DISLOCATION OF THE HIP OCCURRING SIMULTANEOUSLY IN ONE CAR ACCIDENT

By J. C. F. LLOYD WILLIAMSON, Lieut.-Colonel, R.A.M.C.
OFFICER I/C SURGICAL DIVISION OF A GENERAL HOSPITAL

The hip is a strong joint and not dislocated without considerable force applied in the right direction with the thigh in a given position. Not long ago traumatic dislocation of this joint was relatively rare, but the increase in volume and speed of motoring in the last two decades has markedly raised the incidence. Fast-moving, mechanized warfare has further sharply augmented the number of such cases.

Here is an example typical of many: During the secret flanking move of the 4th Indian Division in Tunisia from the 8th Army, held at Enfidaville, to share in the final break through to Tunis from the West, a tank transporter's brakes failed on a mountain pass. The driver, *faute de mieux*, "took a house" to check his career. When he was dug out from beneath the transporter and house debris, he was found to have a posterior dislocation of the hip. Under difficult circumstances, two attempts were made to reduce this but without success. Finally, 36 hours later, after a truck journey of 150 miles, much of it over mountain tracks, he arrived in the small hours at a 1st Army General Hospital, where, with some effort, reduction was effected.

The writer has seen more dislocated hips during the war years than in twice as many pre-war years, even though these were spent in a region where road accidents were perhaps above the average for the country.

It is, then, an accepted fact that this 'dashboard dislocation' is now the commonest type of traumatic dislocation (Funster et al., 1938). The right is the more frequently injured hip, and posterior dislocation is reported to be seven times as common as anterior (Walker, 1940). The victim, usually the front-seat passenger (Watson-Jones, 1944), is sitting with the hip flexed and adducted. A head-on collision occurs; the knee hits the dashboard, and the head of the femur, which in this position lies almost entirely out of the acetabular cup, is forced through the capsule posteriorly. Often this is complicated by a fracture of a greater or lesser portion of the posterior acetabular rim.

The main interest in this report is the triplication of the lesion—three cases from one accident. Cases of bilateral fracture-dislocation have been reported (Gardiner, 1939; Taylor, 1940), but a search, rather superficial owing to the "exigencies of the Service", of the recent literature has failed to disclose so multiple a series of lesions.

CASE REPORTS

On June 29, 1945, a left-hand drive Command car was proceeding along a typical tree-lined *pavé* road near the Franco-Belgian frontier after a shower of rain. Such road surfaces are notoriously skiddy and this was no exception. On a slight bend the car, which was

being driven on the right side of the road, skidded across the carriageway to the left and came to a sudden halt when the middle of the front bumper violently hit a substantial tree on the left of the road.

There were in the vehicle, as is shown in the sketch (*Fig.* 432), three American N.C.O.'s: Cpl. A. (driving), Cpl. U. (passenger on right front seat), and Sgt. D., who was seated on the right back seat with a pile of kit to his left.

The two corporals in front both sustained fracture-dislocations of the right hip: it is suggested that the effect

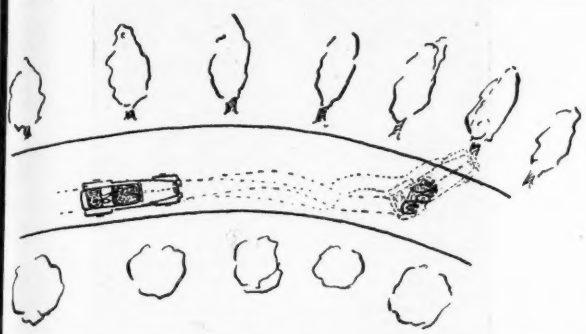


FIG. 432.—Sketch of accident showing relative positions, etc.

of skidding to the left would tend to advance the right side of the body and so the right knee hit the dash-board. Sgt. D., in the back, however, volunteered the remark, that, when he saw the crash was inevitable he half turned to the right to try to get out. As one would expect, his lesion was therefore left-sided. The injuries, as viewed from above, are portrayed in *Fig.* 433.

The accident occurred about 16.40 hr.; first-aid was rendered, and the cases reached hospital at 19.15 hr. and were at once examined. Their further histories were as follows:—

Cpl. A. (aged 22).—Was first examined: he was clearly moribund, his main lesion being a fracture of

FIG. 434.—*Cpl. A.* Unreduced presumably posterior dislocation of right hip. A fairly large segment of acetabulum has been fractured and carried upwards with the head.

the skull, probably of both base and vault. A cursory general examination, however, showed the typical deformity of a posterior dislocation of the right hip. He died at 19.55 hr. The report of a single antero-posterior radiograph (*Fig.* 434) taken post mortem was: "Upward and presumably backward fracture-dislocation of the right hip. A fairly large segment of the acetabular rim was carried upwards with the head."

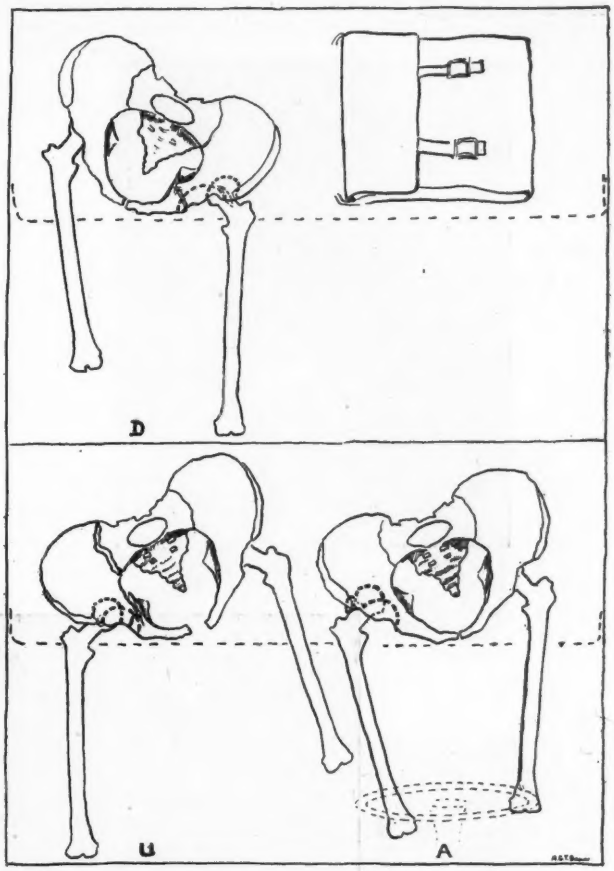


FIG. 433.—Sketch of pelves immediately after impact (seen from above).

Cpl. U. (aged 23).—Cpl. U. was then examined. He was rather cold and restless, but his general condition appeared surprisingly good when one contemplates the multiplicity of his lesions:—

1. Open comminuted fracture of right tibia and fibula (large open wound of mid leg—tibia broken into three equal thirds).
2. Fracture-dislocation of right hip (posterior dislocation with fracture of thin fragment of rim) (*Fig.* 435, A).
3. Fracture of pelvis (from right acetabulum obliquely backwards above tuber ischii).
4. Disruption of pelvis (wide separation of symphysis pubis and splaying of right sacro-iliac joint) (*Fig.* 435, B).
5. Closed fracture-dislocation of left ankle (1 in. lateral displacement of foot).
6. Lacerations of face and left elbow.

Resuscitation was ordered together with penicillin (systemic course started—total 1,001,000 units), tetanus toxoid, etc.

At 20.45 hr. he was moved to the X-ray department and thence to the theatre at 21.15 hr.

OPERATION.—Under pentothal 1 g. and oxygen anæsthesia (Major J. V. Broad, R.A.M.C.).

The lesions were tackled *seriatim*:—

1. Toilet of open fracture of right leg.
2. Reduction and plaster of fracture-dislocation of left ankle.

3. Patient moved to mattress on floor and with assistant steadying lightly bandaged right leg, the dislocation of right hip reduced without difficulty by 'lifting' head into acetabulum.

4. Application of plaster (padded and split) on right leg fractures with a slight manual traction—from base of toes to mid-thigh.

"Left ankle: Almost perfect reduction—slight residual lateral subluxation."

Further progress was uneventful except for urinary retention, which once required catheterization. The right lower limb was partially immobilized by sandbags, but in view of the multiplicity of injuries, no attempt was made either to extend limb or to nurse patient on

A

C

B

FIG. 435.—*Cpl. U.* A, *Before manipulation.* Comminuted fracture-dislocation of right hip. Femoral head displaced upwards and backwards. Thin fragment of rim of acetabulum (approx. 1 in. long) detached posteriorly and displaced with head. Also oblique fracture through acetabulum. B, *Full pelvis after manipulation.* Showing wide separation of symphysis pubis and splaying of right sacro-iliac joint. C, *After manipulation.* Reduction of dislocation complete. Rim fragment still displaced.

5. Toilet and suture of lacerations of eyebrows and elbow.

6. Passing of catheter—clear urine withdrawn.

Plasma infusion (2 bottles) given during operation, which took 90 minutes. Condition at end satisfactory.

Post-operative radiographs were taken later:—

"Right hip: Reduction of femoral head complete. The rim fragment remains widely displaced" (*Fig.* 435, C).

"Right leg: General alinement of fragments surprisingly good under circumstances: some angulation."

his side or in sling to allow pelvis to close. For administrative reasons, he and Sgt. D. were transferred on July 8 (the ninth day) to a U.S. General Hospital.

SUBSEQUENT HISTORY.—On inquiry on July 24 we learnt that "progress was excellent". Os calcis pin traction to right lower limb and pelvic slinging (with partial reduction of spread of pelvis) had recently been instituted.

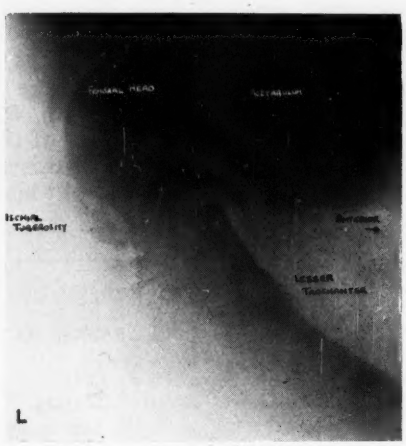
Sgt. D. (aged 25).—This man was last examined. He was in good general physical condition, but more

"hit up". There was the typical deformity of a posterior dislocation of the left hip and internal rotation and adduction of hip with flexion of knee and hip; in addition he showed marked swelling of the right ankle. This, however, proved to be a very severe sprain with no bony lesion. He was X-rayed: "*Left hip*: Comminuted fracture-dislocation. Head dislocated upwards and backwards. A segment (at least one-quarter of rim) of acetabulum detached posteriorly and displaced upwards with femoral head. Also linear fracture from acetabulum to tip of tuber ischii" (*Figs.* 436, A, B).

At 23.30 hr., under pentothal (g. 1) anæsthesia, this left dislocation was reduced as in the previous case, and fixed Thomas splint strapping extension applied; the right ankle was immobilized in a padded plaster.

Post-operative radiographs confirmed the reduction of the dislocation, but the acetabular fragment, although nearer, remained widely displaced and at right angles to its true position (*Figs.* 436, C, D). In this case again there was temporary dysuria and catheterization was necessary once. There was some pyrexia for a few days, probably due to absorption from hæmatomata of left thigh and right ankle, but this did not appear in any way to affect his general condition, which remained good throughout. He was transferred on July 8.

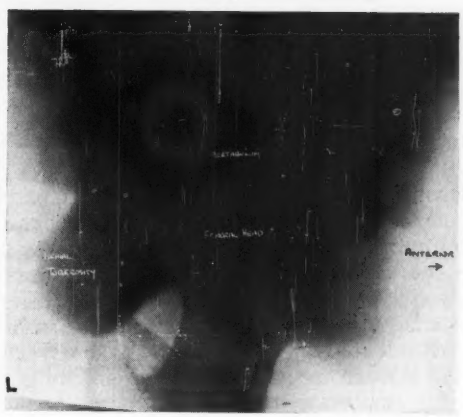
SUBSEQUENT HISTORY.—By July 24 it was reported that the displaced acetabular fragment had been removed operatively. Traction had been maintained and it was proposed shortly to apply a plaster spica. "Convalescence uneventful."



A

B

C



D

FIG. 436.—*Sgt. D.* A, B, *Before manipulation.* Comminuted fracture-dislocation of left hip, femoral head displaced upwards and backwards. A segment, which includes at least one-quarter of rim of acetabulum, has been fractured and displaced with head. Also fracture through acetabulum and tuber ischii. C, D, *After manipulation.* Reduction of dislocation complete. Rim fragment still widely displaced and at right angles to its normal position.

DISCUSSION

The concomitant occurrence of a fracture of the acetabular ring increases the severity of the lesion and makes the ultimate prognosis of dislocated hip less hopeful.

More often than not the displaced fragment follows the head on reduction and is replaced into normal or almost normal position. In these cases this did not transpire. In the case of Cpl. U., in view of the small size of the fragment and the presence of the other injuries, no further attempt

was made to replace the rim fragment, but Sgt. D.'s larger fragment was removed.

To lessen the risk of redislocation, it is more usual to attempt replacement rather than removal, but the operation was carried out elsewhere and one cannot tell what was actually found at operation.

The damage to the acetabular ring by fracture renders the hip potentially unstable, and it is important to maintain traction for four to six weeks and immobilization for a yet longer period. These measures also lessen the risk of traumatic myositis ossificans, avascular necrosis of the femoral head, and osteo-arthritis : all these not infrequently follow this type of injury.

There was no evidence of any sciatic nerve palsy in either case.

The force applied to the pelvis was so severe that it not only produced a fracture-dislocation of the hip and split the pelvis from acetabulum to tuber ischii in the second and third cases, but in the second also disrupted the pelvic ring ; it is not, then, surprising that micturition was temporarily disturbed. Separation of the symphysis pubis is frequently associated with extraperitoneal rupture of the bladder.

The pelvic lesion of the driver (Cpl. A.) was the least serious of the three. The grip on the steering wheel is thought to have checked the momentum of the driver and so lessened the force of his impact, but unfortunately his head also hit something, probably the tree, with fatal results.

SUMMARY

1. A description is given of three cases of fracture-dislocation of the hip (all had other injuries in addition) occurring simultaneously in one car accident.

2. The condition is briefly discussed.

I wish to express my thanks to Col. L. Handy, A.M.S. (Commanding Officer of the General Hospital), to Major D. W. Seth Smith, R.A.M.C., and Sergt. H. A. Burgess, R.A.M.C., of the Radiological Department for their skilled assistance, and to Capt. A. G. T. Brown, R.A.M.C., for his excellent sketches.

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SIMULTANEOUS CARCINOMA AND TUBERCULOSIS OF THE COLON

REPORT OF A CASE AND REVIEW OF THE LITERATURE

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THE association of active tuberculosis with carcinoma has been recorded in most of the organs of the body. The commonest sites are the skin, larynx, and lung, where the development of cancer is commonly a complication of chronic tuberculous ulceration. The stomach, colon, uterus, and testicle, which are less often infected by tuberculosis, have nevertheless, provided numerous instances of this double pathology. In reviewing the colonic cases the writers were struck by the preponderance of ileocæcal cases. As the ileocæcal region is by far the commonest site for colonic tuberculosis, the frequent association of the two conditions here seems to indicate that the tuberculosis precedes the carcinoma. It could be argued, on the other hand, that the massive type of carcinoma commonly found in the cæcum is more likely to provide a suitable nidus for the growth of ingested tubercle bacilli than the relatively smaller and less necrotic cancers which occur in the right colon. Our case is of interest in being the only recorded instance of this association in the region of the splenic flexure, which is a very rare site for colonic tuberculosis.

The coexistence of the two diseases seems to have been first reported by Bayle in 1810. Rokitansky in 1854 taught that the two diseases were antagonistic, a view which has had its proponents and antagonists up to the present time. Thus Lubarsch in 1888 found among 2668 cases of tuberculosis 117 (4·4 per cent) with associated carcinoma, and among 569 cases of carcinoma 117 (20·6 per cent) with tuberculosis. Of 5967 cases without carcinoma 2251 (42·7 per cent) were tuberculous. These figures indicate that carcinoma is twice as frequent in non-tuberculous patients and that tuberculosis is twice as frequent in non-carcinomatous patients. More recently, Pearl (1929) studied the association of the two diseases in 7500 autopsies and concluded that the antagonism was sufficient to support the treatment of cancerous patients by tuberculin.

The opposite view was well expressed by Carlson and Bell (1929), who, after studying 1,195 cases, reported Pearl's conclusions unjustified and remarked that the only way in which active tuberculosis can be said to prevent cancer is by killing the patient before it develops. Cooper (1929) analysed 247 cases in which the two diseases occurred in the same organ and concluded that their simultaneous occurrence was sufficiently frequent to disprove a mutual antagonism. Among his collected cases are 48 in the gastro-intestinal tract, of which 23 were in the

large intestine. One other case has been recorded in the literature since his paper in addition to the one reported below.

CASE REPORT

Mrs. H. E., a married woman, aged 67, was admitted to hospital on April 12, 1945, for recurrent attacks of abdominal pain, diarrhœa, and vomiting.

HISTORY.—As the patient was confused and garrulous this was unreliable. She had noticed an abdominal tumour for some months, had felt weak and ill for the same time, and about three weeks before admission began to suffer from recurrent attacks of colic and vomiting, followed by diarrhœa. These attacks lasted for a day or two and were accompanied by abdominal distension. Her appetite was poor and the stools were normal in appearance, without overt blood or mucus.

ON EXAMINATION.—She was slightly confused mentally, and appeared moderately anæmic. Her pulse was 88, temperature 98°, and blood-pressure 130/75. There were no abnormal signs in the chest, and this was normal on X-ray examination. The abdomen was distended and there was a hard irregular mass occupying most of the left hypochondrium, flank, and iliac fossa. It could not be moved independently of the anterior abdominal wall, but was not fixed posteriorly. The liver was not enlarged. Rectal examination was normal and there were no signs of free fluid in the abdomen.

Investigations.—A barium enema showed some narrowing of the pelvic and splenic flexure regions of the colon, but the barium flowed quite freely past the narrowings. The appearances were rather suggestive of external pressure.

The blood-sedimentation rate was 25 mm. in an hour (Cutler's method).

Red blood-count 4,550,000 per c.mm.

Hæmoglobin 64 per cent Haldane. Colour index 0·71.

Total white count 8000 per c.mm. Differential count within normal limits.

A provisional diagnosis of either a colonic or ovarian carcinoma was made and the patient operated on after a week's preparation.

AT OPERATION.—The abdomen was opened through a left paramedian incision. As the rectus sheath was opened it became apparent that the muscle and underlying peritoneum were being infiltrated by the growth. A part of the muscle and peritoneum was therefore left attached to the tumour and eventually removed with it. The growth was found to be arising from the left end of the transverse colon, where it was attached to the greater curvature of the stomach. It extended downwards in front of the descending colon, to which it was also attached. There were no secondary deposits in the liver; the mesocolic glands could not be examined at this stage, owing to the large mass in front of them. The descending colon and splenic flexure were mobilized, the iliac and transverse colons divided, and the greater curvature of the stomach resected. The growth was now free and was liberated by dividing the left part of the transverse mesocolon. At this stage glands were discovered in the root of the mesocolon which could not be removed entirely owing to their relation to the superior mesenteric artery. One of these was removed separately and was later proved to be tuberculous.

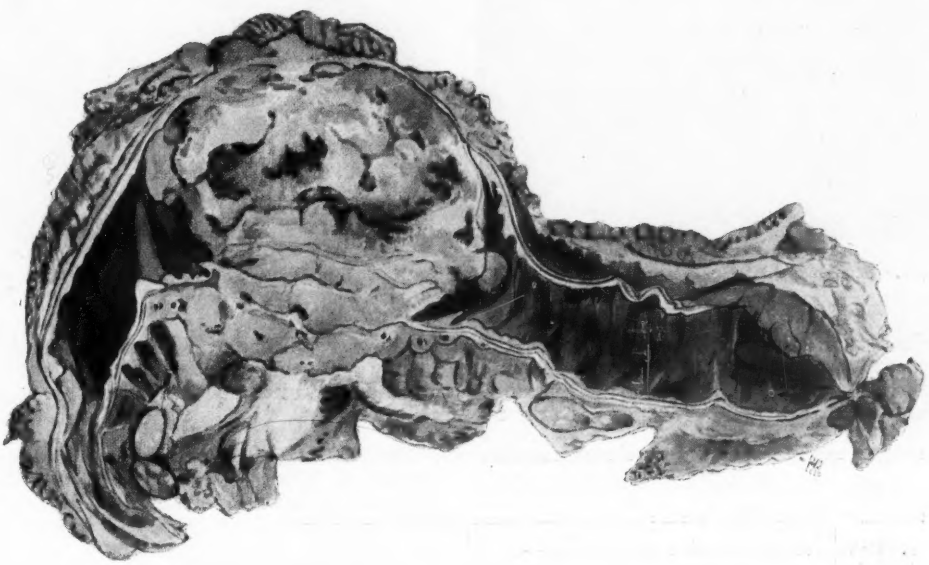

FIG. 437.—Drawing of tumour.

The transverse and iliac colons were joined by end-to-end anastomosis and the stomach repaired. The abdomen was closed with some difficulty owing to the defect left by the removal of a considerable part of the rectus muscle and peritoneum.

The patient stood this operation very well and was returned to her bed in good condition.

POST-OPERATIVE PROGRESS.—This was uneventful until the eleventh day, when the wound burst open in the centre and discharged a little pus. The dehiscence, which involved skin only, slowly spread up and down, until about one month after the operation a typical tuberculous ulcer, about three and a half inches long and half an inch across involved the greater part of the scar. This was excised by diathermy and the floor curetted vigorously. The response to this treatment was most satisfactory, vigorous red granulation tissue formed within a week, and the wound is now (three months post-operative) almost completely healed.

DESCRIPTION OF THE TUMOUR.—The tumour (*Fig.* 437) measured about 6 in. in its largest diameter and had portions of the rectus muscle and stomach attached to its surface. On section the centre of the mass presented the typical appearance of a massive carcinoma of the colon and was of a greyish colour, flecked with

hæmorrhages. The peripheral part was yellow and had the appearance of a caseating tuberculous granuloma. All the glands sectioned were typically tuberculous to the naked eye.

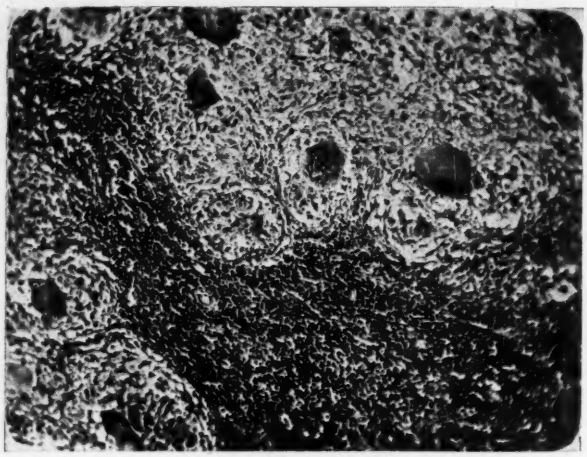

FIG. 438.—Section of gland—tuberculosis only.

Microscopically, the glands showed typical caseating tuberculosis with many Langhans cells (*Fig.* 438). There was no sign of carcinoma in any of the numerous sections which were examined. Sections from the centre of the mass (*Fig.* 439) showed a well-differentiated adenocarcinoma undergoing necrosis. Intimately mixed with the cancer cells was an active tuberculous process, the two lesions being visible in the same microscopical field in several places. The carcinoma did not penetrate deeply and sections taken of the periphery of the mass and the invaded abdominal wall showed tuberculosis only.

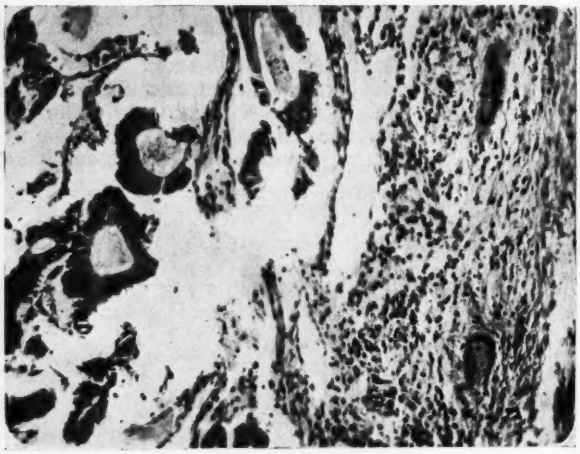

FIG. 439.—Section of tumour—carcinoma and tuberculosis.

DATE	AUTHORITY	AGE AND SEX	SUMMARY
1. Ileocæcal Tuberculosis and Cancer.—			
1897	Naegeli	—	T.B. and colloid cancer of cæcum. T.B. in mesenteric nodes, not in growth.
1897	Metterhausen	35, F.	Adenocarcinoma of ileocæcal region. T.B. granulations invading growth
1900	Crowder	54, F.	Carcinoma of cæcum with tuberculous nodes
1903	Letulle	—	6 cases of carcinoma of appendix in tuberculous patients. In one they were associated in the appendix
1908	Buday	—	T.B. fistulous tract leading to carcinoma of appendix
1910	Stetten	—	T.B. and carcinoma of cæcum
1917	Forman and Cameron	—	Adenocarcinoma of cæcum with tubercles beneath the peritoneum
1921	Herrenschmidt	—	Carcinoma of cæcum. T.B. nodes
1921	Hartmann and Renaud	—	T.B. and carcinoma seen in the same microscopical field of cæcal tumour
1926	Hamperl	51, M.	Adenocarcinoma and T.B. of cæcum
1926	Hamperl	47, F.	Adenocarcinoma and T.B. of cæcum
1927	Hamperl	17, F.	Adenocarcinoma and T.B. of cæcum
1927	Harbitz	30, F. 28, M.	Two cases of associated cancer and T.B. of ileocæcal region. Both diseases intimately mixed
1930	Cooper	44, F.	Adenocarcinoma of ascending colon. T.B. of appendix stump
1930	Cooper	48, M.	Adenocarcinoma of ascending colon. T.B. of mesenteric nodes
1930	Harada	54, F.	Carcinoma and T.B. of cæcum
1936	Hoeffel	42, F.	Carcinoma and T.B. of cæcum causing acute obstruction
1935	Safwenberg	57, M.	Carcinoma and tuberculosis of ascending colon and ileocæcal valve
2. Sigmoid Cancer and Tuberculosis.—			
1902	Moak	—	Adenocarcinoma and T.B. in sigmoid. Metastases of both in the liver and kidney
1903	Guberman	—	Adenocarcinoma of sigmoid with T.B. caseation around the growth
1910	Tauschwitz	36. M.	Both conditions in sigmoid
1917	Reinhardt	69, F.	Stenosing cancer of sigmoid with tubercles in serosa. Older tubercles in lung
1936	Miller	62, M.	Patient died of phthisis. At P.M. mass in descending colon. Mixed tubercle and cancer
3. Rectal Cancer and Tuberculosis.—			
1902	Plien	34, F.	Malignant ulcer of rectum with tubercles within it
1910	Stettin	—	Carcinoma and tuberculous fistula-in-ano
1925	White	—	Carcinoma of upper rectum with metastases in liver; also T.B. in rectum and regional glands.
1937	Uher and Rapant	33, F.	Carcinoma of rectum with typical tubercles between islets of carcinoma.

Tubercle bacilli were seen in Ziehl-Neelsen films of the tumour and the glands.

SUMMARY OF PREVIOUSLY REPORTED CASES

Owing to the inadequacy of some of these reports it is impossible to criticize these cases in detail. It is apparent, however, that in many the condition was cancer of the bowel with tuberculosis of the regional glands, which might well have been of long standing and unrelated to the carcinoma, The cases reported as carcinoma of the appendix might now be regarded as carcinoid tumours.

COMMENTARY

Lubarsch (1888) states that the association of tuberculosis with cancer may occur in any of the following ways :—

1. A chance coincidence without any apparent relation.
2. Metastatic carcinoma developing in an old tuberculous lesion.
3. Secondary infection of carcinoma with tuberculosis.
4. Chronic progressive tubercle in which a carcinoma develops.
5. Simultaneous development of both.

In such a case as ours it is impossible to be certain how the condition arose. The absence of fibrosis in the tuberculous lesion and of old tuberculous glands with calcification makes it improbable that the tuberculous lesion was of long standing, and the absence of a pulmonary lesion with tubercle bacilli in the sputum is against the assumption that a primary carcinoma was secondarily invaded by swallowed tubercle bacilli. Certainly the tuberculous process seems more active than the carcinoma, but that does not prove that it preceded the latter.

SUMMARY

A case of associated active tuberculosis and cancer of the splenic flexure of the colon is described.

The literature is reviewed and the question of the aetiological relationship of the two diseases is discussed briefly.

We have to thank Dr. A. Elliott, County M.O.H., for permission to report this case, which was treated by us at the County Hospital, Farnborough, Kent. Also, Mrs. Helen Randall for the coloured illustration of the specimen, and Mr. J. E. Andrews, of the Southern Group Laboratory, L.C.C., for the microphotographs.

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SOLID TUMOURS OF THE EPIDIDYMIS

REPORT OF TWO CASES

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TUMOURS of the epididymis are still admittedly regarded as rare, yet one of the present writers has in five years seen three such tumours of glandular type, which are similar to one another and which closely resemble those described by Taylor and Davis (1941), and Mackay (1943), and which, moreover, seem related to the tumours described by Halpert (1941), and Friedman and Grayzel (1942). Mackay (1943) observed the essential similarity of the " adenocarcinomata " of Thompson (1936), the " adenomata " of Taylor and Davis (1941), and of Blumer and Edwards (1941), and the " lymphangioma " of the literature ; he himself added two cases. He regarded the tumours as " mesotheliomata ".

Of the three tumours mentioned in the first paragraph, one was described and figured in this JOURNAL by Blumer and Edwards (1941) ; brief accounts of the other two follow.

CASE REPORTS

Case 1.—

CLINICAL DETAILS.—A. W., aged 34. Examined Dec. 11, 1943. Stated he had had painless testicular swelling for some years which had been getting larger recently. A small tumour of lower pole of right testicle.

OPERATION (Jan. 25, 1944).—Exposure of right testicle. A solid tumour of lower pole of epididymis was resected.

FIG. 440.—*Case* 1. Typical tubules. (× 60.)

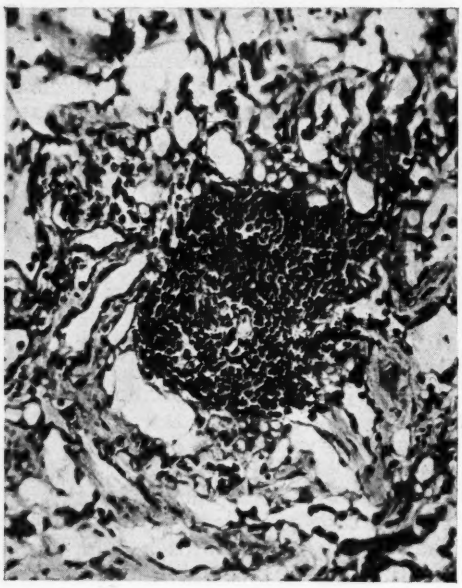

FIG. 441.—*Case* 1. Lymphatic collections. (× 150.

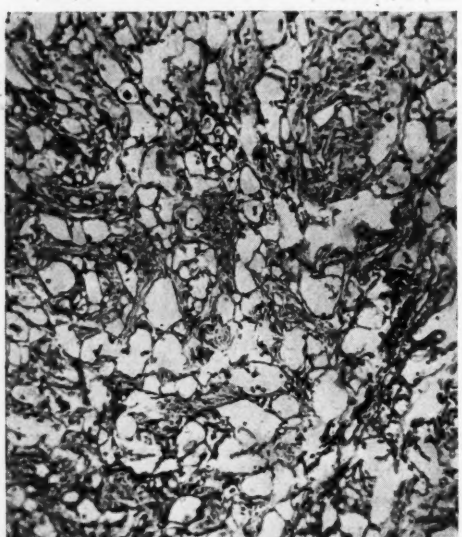

FIG. 442.—*Case* 2. Typical tubules. (× 60.)

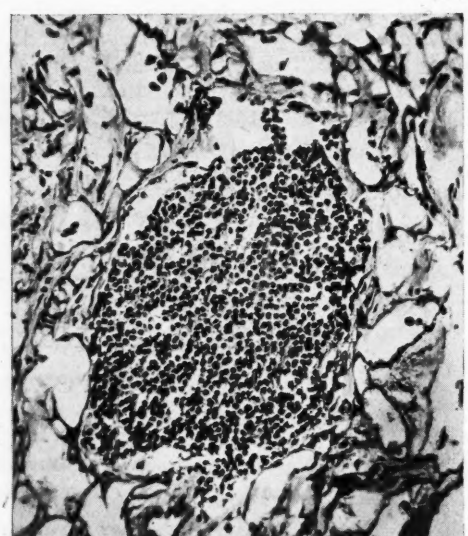

FIG. 443.—*Case* 2. Lymphatic collections. (× 200.)

Case 2.—

CLINICAL DETAILS.—A. J. T., aged 43, a carpenter. On Nov. 10, 1943, he stated that for some months he had had a small swelling on the left side of the scrotum. He had had no pain, but had a certain amount of discomfort. On examination a small firm swelling, the size of a small marble and apparently attached to the globus minor of the left epididymis, was found. The tumour was well defined and obviously not malignant, and it was concluded, in spite of its position, that it was probably a spermatocele. Its removal was advised, but as he was unwilling to submit to this, a suspensory bandage was fitted. In February, 1945, the tumour was slightly larger. Its removal was again advised, and this time he agreed.

OPERATION (April 11).—A left inguinal incision was made and the left testicle exposed. A small hydrocele was present and the tumour in the globus minor. This was removed without difficulty.

The Specimens

These were similar. The first measured approximately 2 cm. × 2·2 cm; the second 1·7 cm. × 1·25 cm. Both were firm to the touch and the cut surfaces yellowish-white in colour.

Microscopical Examination.—Each tumour shows a fibrous capsule containing plain muscle-fibres in varying amount. Within the capsule the tissue consists of tubules lined by a single layer of cells which are somewhat irregular, some flattened, some cubical.. The nuclei are rounded and vesicular. The tubules, though usually empty, in places contain wisps of homogenous pink-staining fluid, and occasional cells, which are probably shed from the walls, are seen. No mitotic figures were seen. Collections of lymphocytes, having the appearance in one or two instances of 'germinal centres', are scattered amongst the tubules. (*Figs.* 440–3.)

The foregoing description would fit both tumours; however, a difference is noticeable in regard to the distribution of plain muscle; in the first, plain muscle is present in the capsule and in the walls of blood-vessels—larger than capillaries—but in addition, substantial bundles of interlacing plain muscle and collagen extend centripetally at one point from the capsule; in the second, plain muscle is seen only in the capsule and vessels.

DISCUSSION

The resemblance between these two tumours and the tumours described as "adenomata" by Taylor and Davis (1941) and Blumer and Edwards (1941) is close. The possible diagnoses are discussed in the latter paper; the most likely would seem to be 'adenoma', 'angioma', or 'endothelioma' (which term is held to include 'mesothelioma'). Mackay (1943), as already mentioned, noted the similarities of the tumours to those described as "adenocarcinomata" by Thompson (1936) and as "lymphangiomata" by various authors. He regarded them as "mesotheliomata", but the presence of plain muscle inside the capsule in our first case seems to provide a transition between 'glandular' tumours and the "mixed leiomyoma and lymphangioma" of Halpert (1941) and the "adenomyoma" of Sakaguchi (1915). Moreover, since the tumour described as "myoma" by Friedman and Grayzel (1942) contained "glandular structures", it seems probable that other 'leiomyomata' contained 'glandular' elements, and were of the same essential nature. Plain muscle is, of course, found in close proximity to epithelial elements in the epididymis.

Several of the 'glandular' tumours, including our two specimens, contain lymphocytic collections showing apparent 'germinal centres'; Thompson (1936) in his review mentioned a "lympho-endothelioma", and Sakaguchi's case (1915) showed lymphoid tissue. The more irregular of the cells lining the tubules resemble somewhat the cells in the sinus of a developing lymph-gland, and in the literature tumours—apparently identical in form, as Mackay (1943) observed—are described as "lymphangiomata"; yet the tubules are almost invariably empty. This seems a difficulty to the present writers. Again, whereas the presence of 'germinal centres' does perhaps speak for such a diagnosis, it is by no means conclusive evidence, as witness, for example, the dense lymphoid masses in adenolymphoma and the remarkable relationship of squamous epithelium to lymphoid tissue in lymphoepithelioma. The nature of these last two tumours is still debated, but no one supposes, it is conceived, that either is a lymphangioma. It must be conceded that the distinction of epithelium from endothelium can be a most embarrassing inquiry. It is here submitted that the morphology of the cells lining the tubules provides inconclusive evidence as between 'endothelioma' (or 'mesothelioma') on the one hand, and 'adenoma' on the other. The wide variations between the cells in individual examples of the 'mixed tumours' of salivary glands will be recalled; yet many authors regard these tumours as adenomata of the salivary glands and the cells as epithelial, e.g., Harvey and others (1938). Lymphoid tissue is occasionally seen in these tumours also.

Since 'glandular elements', plain muscle, and lymphoid tissue with 'germinal centres' are associated in many of the tumours, and since their origins are still debated, perhaps the least objectionable, and most non-committal, designation would be 'mixed tumours' of the epididymis; as in the case of the salivary glands the term is held not to possess the implications of teratoma.

The tumours appear to be benign, and the history often covers many years, but the post-operative period is in most cases not yet long enough to be significant.

SUMMARY

Two, apparently benign 'adenomatous' tumours of the epididymis are described. The relationship of such tumours to 'lymphangioma' and 'myoma' in the same site is discussed.

We wish to thank Mr. T. C. Dodds for the photographs and Mr. W. Blackie for technical assistance.

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CONGENITAL ATRESIA OF THE COMMON BILE-DUCT

REPORT OF A SUCCESSFUL CASE

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THE subject of congenital anomalies of the bile-passages has been fully described in recent years by Ladd and Gross (1941), Donovan (1937), Strauss and Gross (1943), Chesterman (1941), and others, and in reporting this case there is little to add to their description.

Other writers have stressed the importance of searching for other congenital anomalies which are liable to occur in the same subject. The severe pyelitis which complicated our case is probably associated with a congenital anomaly of the urinary tract, and a genito-urinary investigation will be undertaken when the child is a little older.

Laparotomy in this case was performed at the early age of 5 weeks, considerably earlier than in most of the reported cases. This was owing to the recent advances in serology which have made diagnosis from icterus gravis simpler and more certain. As soon as the diagnosis is made and the child is in reasonable condition, the operation should not be delayed. Even at the age of 5 weeks severe changes had taken place in the liver and it is probable that such changes are progressive and tend to become irreversible.

CASE REPORT

R. McQ., male, born on Sept. 20, 1944.

HISTORY.—His birth weight was 7 lb. 12 oz. and the delivery was normal. He was quite well until 15 days old, when jaundice developed and rapidly increased. Stools were slimy and light yellow in colour and the urine became darker. He had been breast fed for the first three weeks and then changed over to half-cream dried milk. The highest weight reached was 8 lb. 1½ oz.

Family History.—The parents and elder sister were alive and well. There was no history of jaundice or of any congenital defect.

ON ADMISSION (Oct. 17).—He was approximately a month old and his weight was 7 lb. 12 oz. Nutrition then seemed fair, but he was deeply jaundiced. The abdomen was distended. The spleen and right kidney were just palpable and there was an umbilical hernia. The urine contained many pus cells, a cloud of albumin, and coliform bacilli, a large quantity of bile-pigments, but no bile-salts.

Blood-count:—

2·9 million red cells.
60 per cent hæmoglobin.
1·0 colour index.
26,000 leucocytes with 77 per cent neutrophils.
No nucleated red cells were seen in the film.
Red blood-cells were of normal fragility.
Kahn reaction was negative.
Van den Bergh positive direct reaction; indirect 16 mg. bilirubin per cent.

Blood grouping showed that both mother and baby belonged to Group O (IV) and were rhesus-positive. Bile-pigments were absent from the fæces.

A diagnosis of congenital atresia of the bile-duct was made and laparotomy decided upon.

For 7 days prior to operation sulphathiazole, 0·125 g. four-hourly for 24 hours, then 0·125 g. six-hourly was given. A specimen of urine just before operation on Oct. 24 showed a few leucocytes, no casts or red blood-cells, and much bile-pigment. The red cells and hæmoglobin were as before, but the leucocytes had increased to 60,000, with 87 per cent polymorphs. During this time and until Oct. 26 he ran a low-grade pyrexia, and on Oct. 24, the day before operation, 5 mg. of 1 : 4 diacetoxy-2-methylnaphthalene in oil were given, and also 150 c.c. of normal saline, subcutaneously.

FIG. 444.—Photograph of patient at 11 months.

OPERATION.—

Anæsthesia (Dr. John Ives): The abdominal wall was infiltrated with ½ per cent procaine, and light open ether administered.

Incision: Upper right paramedian, splitting the rectus muscle.

Findings: The liver was enlarged and firmer than normal. The gall-bladder was not appreciably enlarged, but on dissecting out the common bile-duct this was found to be somewhat dilated (about 3 mm. in diameter) and ceased abruptly just before the upper border of the duodenum was reached. On compression, the gall-bladder emptied and some distension of the blind end of the common duct was produced, indicating patency of the cystic duct. On aspiration the gall-bladder was found to contain very pale bile.

Procedure: Cholecystogastrostomy appeared to be the easiest method of establishing a communication with the alimentary tract. The fundus of the gall-bladder

was therefore freed from the liver and sutured to the stomach for a distance of about 1 cm., using interrupted 000 chromic catgut sutures, placed very close together. Incisions were then made into the gall-bladder and stomach, and the anastomosis completed by a similar row of sutures anteriorly. A second line of sutures was not attempted, owing to the extremely small size of the structures.

POST-OPERATIVE COURSE.—On the day after operation, constipated stools were passed consisting of pure white fæcal matter. Eight days after the operation it was noted that the jaundice was still marked but the urine contained less bile-pigment. The child was running a high temperature and was extremely dehydrated and wasted. The urinary deposit still showed many pus cells.

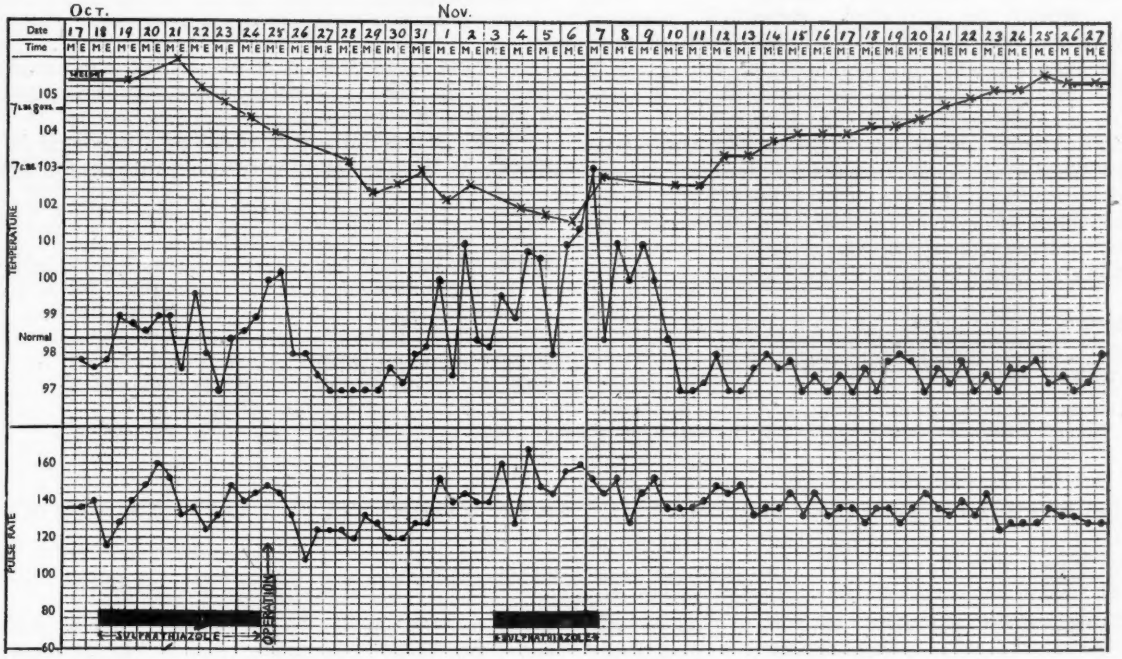

FIG. 445.—Temperature, pulse-rate, and weight chart, Oct. 17–Nov. 27.

The abdomen was closed in layers without drainage. *Operating time*: 40 minutes.

POST-OPERATIVE TREATMENT.—

Days after Operation	*Treatment*
0	250 c.c. of saline intravenously, with 5 per cent glucose, followed by 150 c.c. of Group O stored blood. 1-oz. feeds of 5 per cent dextrimaltose 2-hourly, commencing 4 hours after operation.
1	2-oz. feeds of 5 per cent dextrimaltose 2-hourly.
2	2-oz. feeds of 5 per cent dextrimaltose with ½ drachm half-cream dried milk added.
5	4-oz. feeds containing 2 drachms dextrimaltose and 2 drachms half-cream dried milk 3-hourly.
7	Sulphathiazole commenced (0·25 g. followed by 0·125 g. 4-hourly); 20 c.c. whole blood given intramuscularly.
8	20 c.c. whole blood given intramuscularly. Subcutaneous saline commenced.
9	Subcutaneous saline stopped.
11	Sulphathiazole stopped.
12, 13, 15	275 c.c. plasma, together with an equal amount of half-normal saline given subcutaneously.
21	5-oz feeds containing 2 drachms dextrimaltose and 3 drachms half-cream dried milk 4-hourly.

Eleven days after the operation the stool was first noted to have bile-pigments. The blood-count had improved, there being 3·4 million red cells, and 68 per cent hæmoglobin.

The urinary deposit still showed many pus cells.

Fourteen days after the operation the baby was found to be very much less jaundiced. The stool had much more bile-pigment and the general condition was improved. From that time until his discharge the jaundice grew less, more pigment appeared in the stools, and the urine became clear.

The pyrexia subsided on the sixteenth post-operative day and from then until the thirty-second day there was a steady gain in weight from 6 lb. 12 oz., the lowest weight reached, to 7 lb. 12 oz.

For the next four days there was a fall in weight to 7 lb. 4 oz. associated with mild diarrhœa, which responded to a slight reduction in the strength of the formula.

On his discharge, fifty days after the operation, when he was twelve weeks old, his weight was 8 lb. 12 oz.

DISCUSSION

Differential Diagnosis.—In this case differential diagnosis was not difficult. It was important to exclude icterus gravis neonatorum. The late onset of the jaundice, together with the fact that the mother and child were both rhesus-positive, and the absence of an erythroblastic picture in the blood-film, ruled out this, the most important, alternative. The presence of obstructive jaundice would not be conclusively against this diagnosis, as in certain of the more severe cases of this disease, so great is the

blood destruction that an obstructive jaundice is caused by the blocking of the bile-ducts. Anæmia may occur as a secondary manifestation in any infection, so is not a helpful diagnostic feature. Congenital syphilis would only exceptionally give rise to a severe jaundice; a mild degree of jaundice, coupled with hæmorrhages, a rash, enlarged liver and spleen, and radiological changes in the long bones, would be expected. Acholuric jaundice rarely gives rise to severe jaundice of obstructive type at such an early age, and a mild jaundice with anæmia and increased fragility and micro-spherocytosis, are more commonly found.

Physiological icterus does not really enter into the question as it clears up early and is not severe. Jaundice due to sepsis is uncommon and the severe constitutional disturbance and the presence of bile in the stools would exclude it.

Infective hepatitis has not, so far as we are aware, been reported in so young an infant. The long incubation period would at least make it unlikely to occur before the child was four to six weeks old, unless the mother was also infected, when she would also become jaundiced. The van den Bergh would be biphasic.

Obstruction by a mucus plug can only be diagnosed at operation, as it presents the same clinical picture.

We have recently seen a case of severe jaundice and anæmia associated with intraperitoneal hæmorrhage due to a ruptured hæmangioma of the liver in a neonate.

SUMMARY

A case of congenital atresia of the common bile-duct is described. Cholecystogastrostomy was performed with a successful outcome.

This is the ninth successful case reported in the literature, and apparently the first in this country.

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OSTEOCHONDRITIS DISSECANS AND TORN LATERAL MENISCUS

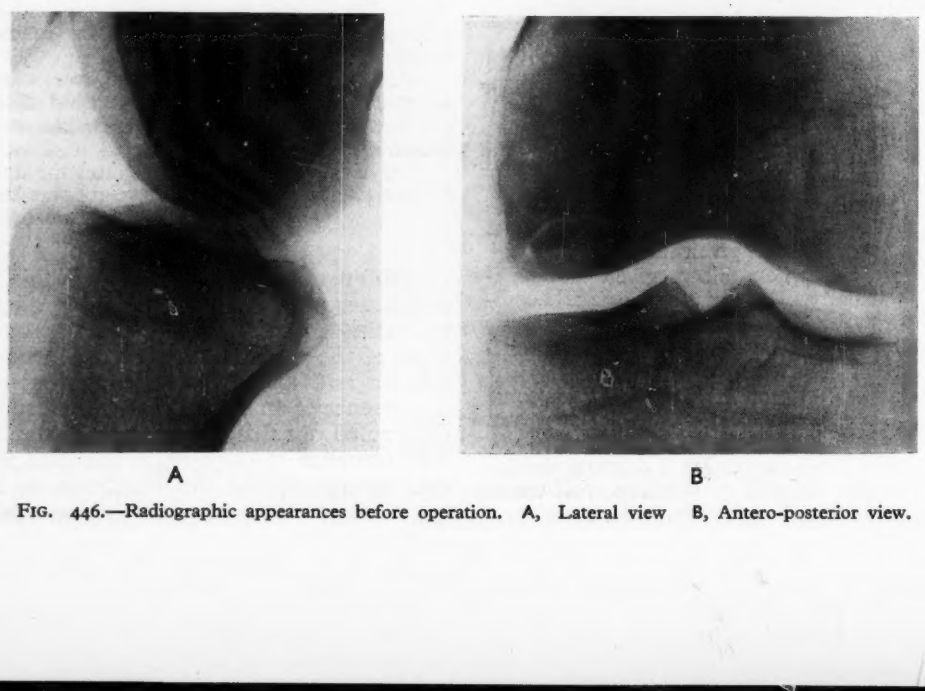
MAJOR J. G. BONNIN, R.A.M.C.

THE absence of universal agreement as to the aetiology of osteochondritis dissecans makes its association with a torn lateral meniscus worth recording. It is to be noted that the lateral condyle, which is the less commonly involved, was affected. There appears to be no doubt in this case that trauma was the aetiological factor, and that the displaced cartilage which followed the injury may have secondarily caused the development of osteochondritis.

CASE REPORT

HISTORY.—E. E., aged 36, a private in the Army, was admitted on account of pain in the right knee, following a long history of attacks of locking. He stated that seven months previously he jumped from a platform and "fell hard". He felt a "jarring" in the knee, and on picking himself up found he was unable to straighten the knee. He was able to walk on the toes. On the following day the knee was slightly flexed but there was little swelling. He hobbled around for six weeks, by which time the knee was almost straight. On one occasion when going up stairs the knee locked and it has locked twice subsequently, the patient being able to release it himself. He complained of increasing pain in the joint, particularly after standing.

ON EXAMINATION.—There was slight wasting of the quadriceps and a small effusion. There was a full range of movement; some tenderness was present over the anterior end of the external cartilage. There was no laxity of any ligament. A clinical diagnosis of torn



A B

FIG. 446.—Radiographic appearances before operation. A, Lateral view B, Antero-posterior view.

lateral meniscus was agreed upon, and a routine radiological examination made. The appearances were as shown in *Fig.* 446, A, B. There was a well-marked osteochondritic focus in the lateral condyle of the femur, with a small dense area surrounded by a zone of decalcification. The osteochondritic fragment had thus not yet separated into the joint. Some suspicion was felt with regard to the diagnosis, but, in consultation with Major-General Max Page, it was decided to explore the joint and to drill the damaged fragment in the hope of revascularizing it and attaching it to its bed.

OPERATION.—Accordingly the joint was opened in the usual manner and the lateral condyle exposed. A curved fissure in the articular cartilage of the femoral condyle was noted, the curve being convex forwards, and the posterior lip being depressed to the depth of the cartilage, in the same manner as a depressed fracture of the cranial vault. On inspecting the cartilage, a complete bucket-handle tear of the cartilage was seen lying turned over on itself in the midline of the joint (*Fig.* 447). The

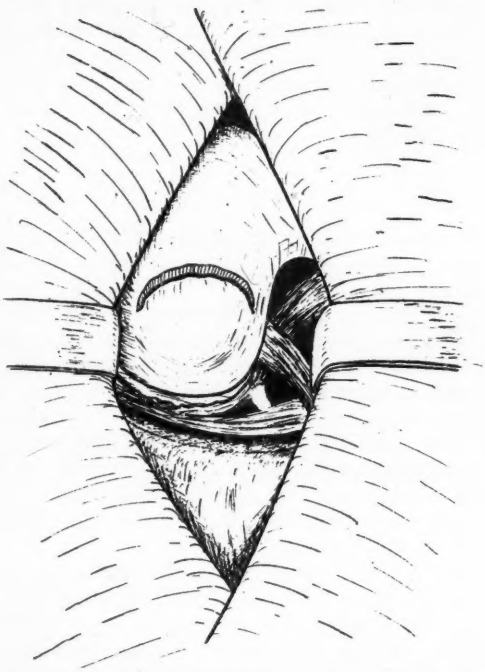


FIG. 447.—The appearance at operation, showing the right lateral meniscus detached from the periphery and turned into the middle of the joint.

complete congruity of the curve of the displaced cartilage and the cartilaginous fracture on the condyle was at once apparent, and on extending the knee the cartilage was found to fit over the depressed fragment and to depress it still further. The cartilage was detached from its anterior attachment and removed completely (*Fig.* 448). Fifteen holes with a $\frac{1}{64}$-in. drill were made through the depressed cartilaginous area into the condyle of the femur, and the joint was closed.

PROGRESS.—Little reaction followed—early movements were prohibited and at the end of a fortnight a knee-fixation plaster was applied and the patient allowed limited activity in it. Six weeks later the plaster was removed. The radiological appearances then were an apparent increased density of the bony fragment due to general decalcification and it was slightly rotated, suggesting that it was lying loose under the cartilage, but the articular cartilage over it appeared whole, while the fragment was slightly reduced in size. There was no effusion.

With the commencement of active movements the effusion recurred. A full range of movements returned in three weeks.

Ten weeks from the removal of the plaster there was still a small effusion, the patient could walk four miles comfortably, and had no complaints.

He was transferred to a Convalescent Depot and returned to duty in category B 7.

FIG. 448.—Photograph of the lateral meniscus, removed at operation, lying in the position it occupied in the joint.

DISCUSSION

No claim can be made as to the effectiveness of the operation. The clinical condition of the knee before and after operation was much the same. The radiological appearances show the fragment to be reduced in size and of similar density, and to be maintaining its attachment to the condyle. The association of osteochondritis dissecans with lesions of the menisci was found in three cases by Fairbank. In two the internal meniscus was damaged, and in one the external meniscus, but in this case the medial condyle was involved. No records could be found of the association of a torn external meniscus and osteochondritis dissecans of the external femoral condyle.

The continued movement of the damaged cartilage on the femoral condyle, as it came in contact with the displaced meniscus, may explain the failure of the bony fragment to redevelop a blood-supply, an hypothesis which has been supported by Timbrell Fisher and Fairbank. It is noteworthy that the lesion is only seen on curved surfaces which are the subject of frictional strain—the capitellum, the upper surface of the talus, and the superior aspect of the femoral head. The occurrence of a subchondral separation under excessive frictional strain could thus be accounted for without the necessity of searching for a prominence against which the condyle impinges. In this case the impingement appears certainly to have been on a displaced meniscus. Whether that alone would give rise to the condition, or whether the continued minor redisplacements of the loose surface cartilage with use, is a necessary additional factor, has yet to be determined. This case supports the traumatic theory of origin suggesting that subchondral fracture, with incomplete separation, followed by continued movement of the damaged surface, produces the typical changes within a period of six months.

SUTURE OF THE EXTERNAL AND INTERNAL POPLITEAL NERVES

By R. ROAF

SURGEON TO A PERIPHERAL NERVE UNIT, EMERGENCY MEDICAL SERVICE

In an ideal nerve suture, healthy axons with their surrounding Schwann sheaths are approximated without tension to the corresponding distal Schwann tubes. In dealing with war wounds such ideal conditions are rarely attained. Accordingly, the more important factors which may impair the result are listed below.

1. Extensive destruction of the nerve, causing undue tension (Highet and Holmes, 1943; and Highet and Sanders, 1943).

2. Extensive fibrosis and degeneration of the nerve consequent upon either infection and/or ischæmia; here again the necessary resection of abnormal tissue may lead to too large a gap and excessive tension, or alternately the mobilization of the nerve necessary to bridge the gap may interfere with its blood-supply. A number of workers (for summary, *see* Adams, 1943) have investigated the blood-supply of peripheral nerves. It seems agreed that most nerves have both a longitudinal and segmental blood-supply which anastomoses freely and that in the experimental animal both supplies must be occluded before ischæmic changes occur in the nerve. This is prima facie evidence that wide mobilization of a nerve may be performed without impairing the blood-supply, and clinical experience confirms this. Nevertheless, some nerves, e.g., the ulnar and external popliteal, seem to be less generously supplied at certain levels and, especially with extensive war wounds, wide mobilization seems to impair further an already scarcely adequate blood-supply.

3. With the mere passage of time the distal Schwann tubes may shrink, thus preventing regeneration of the larger axons (Holmes and Young, 1942; Guttman and Sanders, 1943).

4. The sensory and motor end-organs may atrophy. This will depend on a number of factors —length of time before re-innervation, associated tissue damage, ischæmia, quality and quantity of physiotherapy, etc. It will be noticed that the passage of time may operate in a number of ways: it may indicate infection, extensive tissue destruction, ununited fracture or ischæmia, or it may act *per se* by causing degenerative changes in the nerve or end organs.

5. Height of lesion. It is obvious that after a high suture it will take longer for peripheral structures to be re-innervated; during the interval various degenerative changes occur (*see above*); also it is probable that the rate of axonal regeneration becomes progressively slower with the passage of time (Seddon, Medawer, and Smith, 1943). Also a special factor may be operating, e.g., important blood-vessels may enter the nerve at a certain level; a lesion at this level would be likely to cause nerve ischæmia.

6. The inherent powers of axon regeneration may vary with both the age and general condition of the patient and also with the proximity of the lesion to the nerve-cell. In this survey the first two variables have been largely eliminated. All cases with impaired general health have been excluded, and the great majority of cases have been healthy males between 18 and 30 years of age.

Finally, the degree of successful apposition may vary. Even when every care is taken to suture the nerve-sheath only, to minimize fibrosis and scar tissue, to tie the sutures with correct tension and to orientate the nerve-ends in relation to each other, it is obvious that perfect apposition of each proximal and distal axon sheath cannot be obtained and that there must be considerable 'shunting' of fibres at the suture line. Indeed, it is remarkable that sutured nerves regenerate as well as they do; presumably axonal branching and 're-education' play an important part.

RESULTS

The results of suture of internal and external popliteal nerves have been chosen for three reasons:—

1. The criteria of recovery are simple. On the motor side it is easier to grade a muscle accurately in the leg than in the arm. Owing to overlap and the complex clinical syndromes that appear, it is harder to express sensory recovery in clear-cut grades; in addition, sensory recovery is unimportant in the external popliteal distribution and the finer modalities of sensation are less important in the foot than the hand, so I have omitted sensory recovery in this series.

2. During its course down the thigh, the sciatic nerve only gives off one relatively unimportant branch. Accordingly it is easier to compare the results of 'high' and 'low' lesions than it is to compare above- and below-elbow lesions in the arm.

3. There is a marked difference in results between the external and internal popliteal nerves (*Tables I, II*).

The mode of assessment has been purely clinical. In no case in this series have the distal muscles regained voluntary contraction; accordingly all recoveries would fall into Group M (1) in the Medical Research Council Scheme of grading; but within this group there are important differences which I have classified in the subgroups 'slight', 'fair', and 'good'.

'Slight' implies a voluntary contraction which is not strong enough to produce movement.

'Fair' implies that the muscles can produce movement.

'Good' implies that the external popliteal muscles can dorsiflex and evert the foot against gravity and that the patient can dispense with all appliances. In the case of the internal popliteal muscles it implies that the patient can stand on tip-toe.

Table I.—Results of External Popliteal Nerve Suture (59 *cases*)

Case	Level of Lesion	Gap between Nerve-ends after Resection	Delay between Injury and Suture	Result
		cm.	mths.	
+ L. B.	Middle	5·5	7	Fair
E. B.	Popliteal	8·0	14	Slight
H. B.	Popliteal	?	1	Slight
E. B.	Popliteal	6·5	10	Nil
+ T. B.	Upper	10·0	12	Nil
R. A. B.	Lower	?	7	Nil
+ J. W. B.	Lower	12·5	5	Nil
+ J. B.	Middle	7·0	3	Nil
+ G. C.	Upper	?	2	Nil
+ L. C.	Middle	7·5	4	Fair
E. C.	Lower	5·0	2	Fair
+ J. C. C.	Lower	6·0	4	Fair
G. D.	Upper	7·0	5	Nil
J. D.	Upper	3·0	12	Nil
+ A. C. F.	Upper	8·0	7	Nil
K. F.	Upper	7·0	5	Slight
+ K. G.	Upper	4·0	2	Nil
+ F. G.	Lower	6·0	6	Nil
J. G.	Lower	6·5	6	Slight
J. G.	Popliteal	5·5	6	Slight
W. G.	Upper	5·5	10	Nil
+ E. H.	Middle	7·0	6	Nil
J. H.	Popliteal	6·0	10	Nil
C. H.	Popliteal	5·5	8	Fair
W. H.	Middle	5·5	9	Nil
L. H.	Popliteal	5·5	3	Good
+ D. G. J.	Upper	7·0	5	Nil
W. N. J.	Lower	4·0	3	Fair
S. K.	Popliteal	4·5	6	Fair
P. L.	Upper	5·0	6	Nil
L. L.	Lower	10·0	7	Nil
J. M.	Middle	4·0	5	Nil
L. McD.	Middle	7·0	9	Slight
+ G. McL.	Middle	6·25	7	Slight
J. M.	Popliteal	5·0	7	Fair
+ J. McD.	Upper	6·0	2	Nil
J. F. M.	Lower	7·0	6	Nil
F. N.	Lower	2·5	3	Fair
+ A. O.	Upper	7·5	9	Nil
J. P.	Lower	5·0	4	Nil
T. A. P.	Lower	5·0	10	Slight
R. P.	Lower	3·75	7	Fair
J. R.	Upper	7·0	9	Nil
H. R.	Middle	5·0	9	Nil

'Upper', 'Middle', 'Lower', and 'Popliteal' stand for upper, middle, and lower thirds of thigh and the popliteal space.

From the list of possible variables, three factors have been selected—namely, height of suture, delay in suture, and length of resection. Other factors such as degree of apposition, amount of physiotherapy, the patient's adaptability and powers of re-education, have not easily lent themselves to measurement.

DISCUSSION

From *Table III* it will be seen that in the case of the external popliteal, the low sutures give much better results than the high ones. Indeed, all the 'good' results are from sutures performed in the popliteal space, all the 'fair' results are from sutures in the lower third of the thigh or below except for two, in both of which the length of nerve resected was very small.

From *Table IV* it will be seen that there is little if any difference in the results of suture of the internal popliteal nerve at different levels.

Table V shows that an excessive gap in the nerve is an adverse factor; the results of 'less than 6 cm.' resections are distinctly better than '6 cm. or over.' All fair and good results occurred following less than 6 cm. resection with the exception of three, which were all very low in the thigh.

Table VI expresses the same fact in a slightly different way.

Tables VII and *VIII*, which give comparable figures for the internal popliteal nerve, give similar results but much less marked.

Table IX gives the average delay between injury and suture, and shows that the better results are

Table I.—continued.

Case	Level of Lesion	Gap between Nerve-ends after Resection	Delay between Injury and Suture	Result
J. R.	Popliteal	2·5	5	Good
T. R.	Lower	4·5	8	Nil
J. S.	Popliteal	?	6	Good
H. S.	Popliteal	3·0	6	Good
+ W. E. S.	Lower	?	2	Fair
W. S.	Upper	7·5	13	Slight
H. T.	Middle	11·0	4	Nil
T. T.	Popliteal	6·5	8	Nil
+ W. T.	Upper	4·5	6	Fair
W. T.	Lower	7·0	6	Nil
L. W.	Upper	4·0	7	Nil
+ T. W.	Lower	8·7	8	Fair
+ A. W.	Upper	9·0	5	Nil
F. W.	Middle	5·5	3	Nil
+ H. W. B. Y.	Middle	?	4	Slight

+ indicates a combined lesion of both nerves.

Table II.—Results of Internal Popliteal Nerve Suture (31 *cases*)

Case	Level of Lesion	Gap between Nerve-ends after Resection	Delay between Injury and Suture	Result
		cm.	mths.	
R. A.	Middle	9·0	7	Nil
+ L. B.	Middle	5·5	7	Fair
+ T. B.	Upper	10·0	12	Nil
+ J. W. B.	Lower	12·5	5	Fair
+ J. B.	Middle	8·0	3	Nil
+ G. C.	Upper	?	2	Fair
+ L. C.	Middle	11·0	4	Nil
+ J. C. C.	Lower	5·0	4	Fair
T. D.	Popliteal	8·0	3	Nil
+ A. C. F.	Upper	7·0	7	Nil
+ F. G.	Lower	8·0	6	Slight
+ K. G.	Upper	4·0	2	Nil
+ E. H.	Middle	7·0	6	Fair
+ D. G. J.	Upper	10·5	6	Nil
+ J. McD.	Upper	6·0	2	Fair
+ G. McL.	Middle	6·25	5	Good
V. A. M.	Upper	8·0	7	Nil
D. O'D.	Lower	5·5	10	Slight
+ A. O.	Upper	7·5	9	Slight
F. S.	Middle	5·5	6	Fair
+ W. E. S.	Lower	?	2	Good
T. S.	Middle	6·0	6	Nil
W. S.	Upper	7·5	13	Fair
R. T.	Upper	3·5	2	Fair
H. T.	Middle	11·0	4	Slight
G. W.	Middle	9·0	6	Fair
L. W.	Upper	4·0	8	Nil
+ A. W.	Upper	9·0	5	Fair
+ T. W.	Lower	6·0	8	Fair
+ H W. B. Y.	Middle	?	4	Fair
+ W. T.	Lower	8·5	9	Nil

+ indicates a combined lesion of both nerves.

Table III.—Analysis of Results for External Popliteal Nerve Classified according to Level of Lesion

Level		*Results*
Upper	17 cases	14 nil, 2 slight, 1 fair*
Middle	12 cases	7 nil, 3 slight, 2 fair
Lower	17 cases	8 nil, 2 slight, 7 fair
Popliteal	13 cases	3 nil, 3 slight, 3 fair, 4 good

* Gap of only 2·5 cm.

Table IV.—Analysis of Results for Internal Popliteal Nerve Classified according to Level of Lesion

Level		*Results*
Upper	12 cases	6 nil, 1 slight, 5 fair
Middle	11 cases	4 nil, 1 slight, 5 fair, 1 good
Lower	7 cases	1 nil, 2 slight, 3 fair, 1 good
Popliteal	1 case	1 nil (large gap)

Table V.—Results for External Popliteal Nerve Classified according to Gap after Resection (53 *cases*)

6 cm. or over 27 = 18 nil, 6 slight, 3 fair
Under 6 cm. 26 = 12 nil, 2 slight, 9 fair, 3 good

Table VI.—Relation in External Popliteal Nerve of Result and Gap after Resection

Average length of resection for *all cases* (53) = 6·1 cm.
Average length of resection for 'nil' (30) = 6·6 cm.
Average length of resection for 'slight' (8) = 6·6 cm.
Average length of resection for 'Fair' (12) = 5·2 cm.
Average length of resection for 'good' (3) = 3·66 cm.

Table VII.—Results for Internal Popliteal Nerve Classified according to Gap after Resection

6 cm. or over (21) = 10 nil, 3 slight, 7 fair, 1 good
Under 6 cm. (6) = 2 nil, 1 slight, 4 fair

Table VIII.—Relation in Internal Popliteal Nerve of Result and Gap after Resection

Average length of resection for *all cases* (28) = 7·3 cm.
Average length of resection for 'nil' (12) = 7·5 cm.
Average length of resection for 'slight' (4) = 8·0 cm.
Average length of resection for 'fair' (11) = 6·95 cm.
Average length of resection for 'good' (1) = 6·25 cm.

Table IX.—Relation of Delay before Suture to Result in External Popliteal Nerve

Average delay for whole series (59) = 6·2 mths.
Average delay for 'nil' (32) = 6·5 mths.
Average delay for 'slight' (10) = 7·5 mths.
Average delay for 'fair' (13) = 4·7 mths.
Average delay for 'good' (4) = 5 mths.

Table X.—Relation of Delay before Suture to Result in Internal Popliteal Nerve

Average delay for whole series (27) = 5·9 mths.
Average delay for 'nil' (12) = 6·3 mths.
Average delay for 'slight' (4) = 7·2 mths.
Average delay for 'fair' (13) = 5·4 mths.
Average delay for 'good' (2) = 3·5 mths.

associated with a shorter interval. A similar table for the internal popliteal (*Table X*) gives similar results, but less clear-cut.

Table XI shows the result of combined suture of both nerves, and it is obvious that the internal popliteal nerves shows a higher number of recoveries although the conditions were strictly comparable.

Table XI.—Comparison of Results of Internal and External Popliteal Nerves, where Both Nerves have been Sutured Simultaneously

Internal popliteal	7 'nil', 2 'slight', 9 'fair', 2 'good'	20 cases
External popliteal	12 'nil', 2 'slight', 6 'fair', 0 'good'	

There are three observations which have a bearing on this. Firstly, the external popliteal is more vulnerable than the internal; for example, following a through-and-through wound of the thigh passing close to the sciatic nerve, it is common to find a complete lesion of the external popliteal nerve, but practically no damage to the internal popliteal. Secondly, at operation it is common to find extensive atrophy of the external popliteal nerve; thirdly, it is a common observation that the cut ends of this nerve are relatively avascular. It is suggested that the blood-supply is less adequate (Paterson Ross, 1945) and that because of its inherently smaller powers of regeneration the various adverse factors—a high lesion, an extensive gap, or long delay—have a greater effect than in the case of more vascular nerves. A similar comparison may

be made between the median and the ulnar nerves —the latter is often subjected to extensive mobilization and is relatively avascular and often atrophic and yields less good results. It is interesting to note that the relatively vascular median and internal popliteal nerves are more often associated with the syndrome known as causalgia.

SUMMARY

The results of suture of the external and internal popliteal nerves have been analysed.

In the case of the external popliteal, a high lesion, a long delay, and a large gap are considered to be adverse factors. These factors have a less marked effect in the case of the internal popliteal nerve, probably owing to its greater inherent powers of regeneration, which may be related to its greater vascularity.

I would like to thank Professor Platt for advice and criticism; also Messrs. R. Barnes, C. H. Cullen, and A. S. Kerr for allowing me access to cases on which they have operated.

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HÆMATEMESIS FROM EROSION OF THE SPLENIC ARTERY BY PEPTIC ULCERATION

By IAN AIRD

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Of those vessels whose erosion by peptic ulcer may occasion massive hæmatemesis, the splenic artery is the most notorious source of rapidly fatal hæmorrhage. Erosion of the aorta or of the heart leads to even more dramatic hæmorrhage, but bleeding from these so rarely complicates peptic ulceration that they cannot compare in importance with the splenic artery as sources of hæmatemesis. Surgical arrest of hæmorrhage from the splenic artery is possible only if circumstances of time and place combine in a peculiarly favourable way, and if the tempo of the hæmorrhage does not outpace that of blood transfusion and of the preparations for operation.

CASE REPORT

HISTORY.—On the afternoon of July 19, 1945, Dr. A. G. Badenoch was called to a female patient of 56 years who had collapsed after vomiting a pint of coffee-coloured fluid; he transferred her at once to hospital. There was a twenty-year history of dyspepsia—flatulence, acid eructations, and epigastric pain half an hour after food, relieved by alkalis but not by food. A barium series in 1929 had demonstrated a large ulcer on the lesser curvature and posterior wall of the stomach, but surgical advice had not then been sought. In 1939 there had been a slight hæmatemesis treated medically. The present hæmatemesis had been preceded by two weeks of continuous and disabling epigastric pain.

ON ADMISSION.—On arrival in hospital the patient was pallid and was sweating; the pulse-rate was 104, the blood-pressure 90/60, and the hæmoglobin level 70 per cent. The hæmatemesis was then regarded as of only moderate degree. Morphine was prescribed and a modified Meulengracht diet was begun.

Improvement followed rapidly, and at noon next day the patient was entirely comfortable; the pulse-rate and hæmoglobin level were unchanged, and the blood-pressure was 130/80. Two hours later, however, a small quantity of red blood was vomited and operation was therefore planned, in conformity with the opinion that intervention is desirable if a patient over 45 years of age, and known to suffer from a peptic ulcer, bleeds on the day after the commencement of a hæmatemesis. A blood-drip was set up, but as preparations for the operation proceeded, further fresh hæmatemesis occurred. It had been arranged to operate at 5 p.m.; a few minutes before that hour a litre of bright and partly coagulated red blood was vomited. The transfusion rate was accelerated to a rapid continuous flow, but even a rate of 150 c.c. a minute could not apparently keep pace with the speed of blood-loss and the patient rapidly became pulseless.

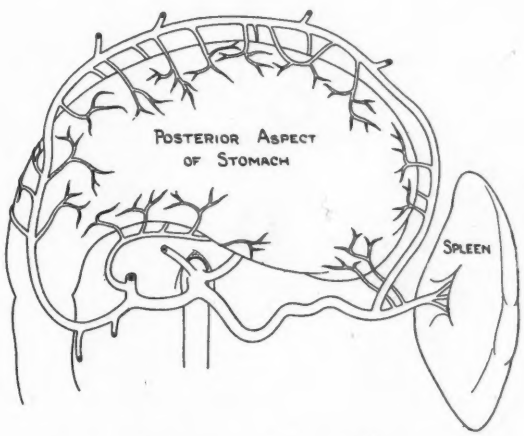

FIG. 449.—Blood-supply of spleen before operation.

FIRST OPERATION (July 19).—Under light gas and oxygen anæsthesia (Dr. F. G. Gibbs) the abdomen was opened by a median supra-umbilical incision. The crater of a large ulcer was felt on the posterior wall of the stomach, midway between the curvatures and a hand's-breadth to the left of the median plane. The stomach was opened between stay sutures in its anterior wall, whereupon some 400 c.c. of bright red blood overflowed from its lumen. The stomach was emptied by suction, but arterial blood spurted from the depths of the ulcer so vigorously that it could not be stayed even

by a thumb thrust into the crater; the ulcer, though palpably fixed to the pancreas, slid away in a curiously elusive fashion between the operator's thumb and the posterior abdominal wall. When, however, a tissue forceps was applied to the gastric mucosa beyond the ulcer and drawn forwards into the wound, the crater was steadied on the tip of the compressing thumb like the apex of a bell-tent on its pole, and bleeding temporarily ceased. At this point a pause was made for blood-replacement to overtake blood-loss.

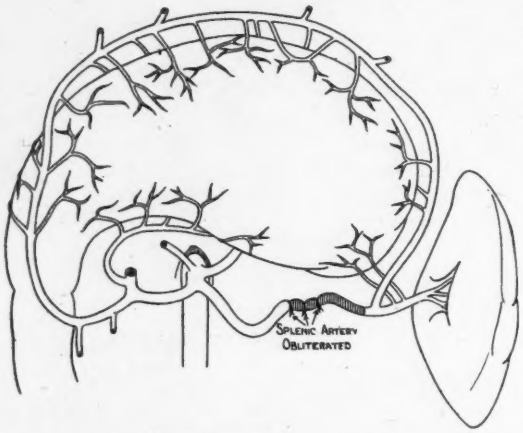

FIG. 450.—Blood-supply of spleen, after obliteration of the main trunk of the splenic artery, showing collateral supply by left gastro-epiploic and short gastric arteries.

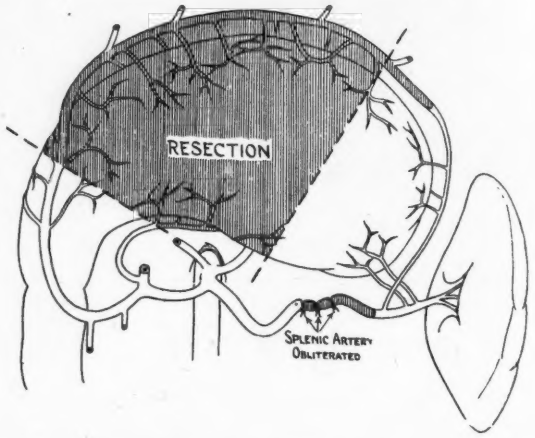

FIG. 451.—Blood-supply of spleen after second operation of gastrectomy, showing dependence on a collateral supply through short gastric branches only.

It was obvious that the source of hæmorrhage was the main trunk of the splenic artery. A series of deep catgut sutures were inserted in the sagittal plane, through stomach wall above and below the ulcer, and through a substantial thickness of pancreas. Only when four of these sutures were tied was the bleeding sufficiently controlled for digital compression to be relaxed. A few further sutures were inserted to obliterate the crater completely, and the stomach and abdomen were closed. It was assumed from the depth of penetration of these sutures that they occluded both splenic artery and splenic vein.

PROGRESS.—Blood transfusion, continued as a steady flow until the bleeding was controlled, was maintained thereafter at forty drops per minute during the succeeding night, to a total amount of three litres. Nasal suction drainage of the stomach and intravenous administration of saline and dextrose solutions were continued for seventy-two hours. The patient's condition improved steadily after operation, no fresh hæmorrhage occurred, and a progressive diet was well sustained after the third day; but the temperature swung daily from 99° to 102° F. for seven days after operation, and for ten days complaint was made of cramping, prostrating, median epigastric pain, greater than any previously produced by the ulcer, and penetrating to the middle of the back. There was no pain in left side or shoulder, and no evidence of pancreatic insufficiency. The patient returned home on the thirty-third day after operation.

SECOND OPERATION.—At subtotal gastrectomy, performed on Oct. 20, the antrum and body of the stomach were removed, together with the vessels on both curvatures. The lesser sac was found almost completely obliterated. The same or a similar ulcer was present on the posterior surface of the stomach, in the same site and of the same size as at the previous operation, but appreciably less deep. As a step in its removal the stomach was separated from the floor of the ulcer by an incision around the circumference of the ulcer, so that the floor was left undisturbed, and a circular defect remained at the ulcer site on the posterior wall of the ablated stomach. The ulcer floor was then seen to be smooth, glistening, and indistinguishable in texture from the neighbouring peritoneum of the posterior abdominal wall. In the substance of the pancreas, deep to the floor of the ulcer, could be seen a white puckered scar, approximately 1·5 cm. in diameter: the sutured ulcer had reopened, but the excavation of the pancreas had healed. The remainder of the pancreas was of a normal appearance and consistence. No pulsation could be felt along the line of the supra-pancreatic portion of the splenic artery, but the spleen was of average size, colour, and consistence, and apparently free from infarction.

The patient's recovery from gastrectomy was uneventful, and again there were no symptoms of splenic infarction; she has now resumed the diet of a healthy person. The platelets on Jan. 2, 1946, numbered 700,000 per c.mm.

SUMMARY AND CONCLUSIONS

1. Hæmorrhage from a splenic artery eroded by a peptic ulcer need not immediately threaten life; as in the case of bleeding from the other arterial trunks which are subject to erosion, a massive hæmorrhage may be heralded by lesser hæmorrhages.

2. A favourable combination of circumstances may permit the surgical control of hæmorrhage from a splenic artery eroded by a peptic ulcer.

3. An intragastric manœuvre is described which facilitates digital control of hæmorrhage from an eroded splenic artery; as soon as digital control is assured, further operative procedures can be suspended until blood-replacement has overtaken blood-loss.

4. Sutures inserted in emergency through the posterior stomach wall in the neighbourhood of an active ulcer, and beyond that deeply into the substance of the pancreas, produce severe post-operative pain; they do not appear to cause pancreatic œdema of a dangerous degree.

5. Obliteration of the splenic artery in its supra-pancreatic course is not necessarily followed by infarction of the spleen: the signs of infarction did not appear even after gastrectomy interrupted the collateral supply along the greater curvature and left the spleen dependent on the short gastric vessels, certainly for arterial supply, and probably for venous drainage as well.

THE SURGICAL TREATMENT OF SEVERE EPISTAXIS IN RELATION TO THE ETHMOIDAL ARTERIES

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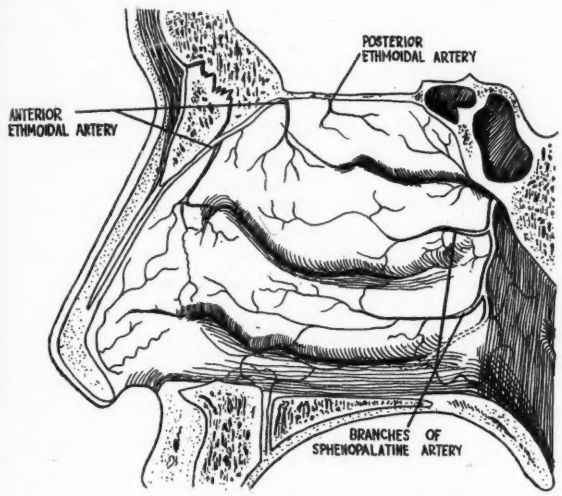
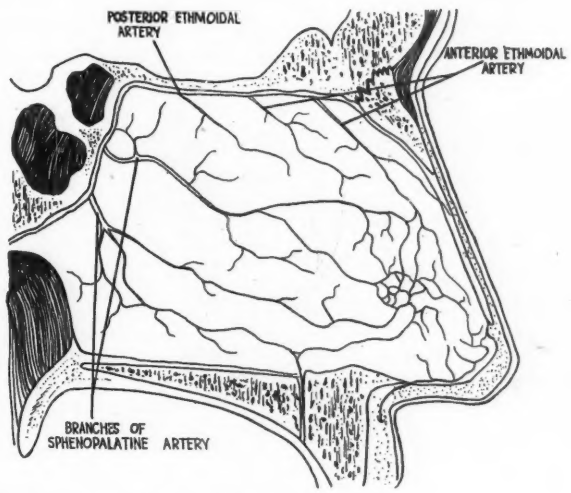
Severe epistaxis is a relatively common emergency, which the otolaryngologist is called upon to treat. Local measures usually suffice to stop the bleeding, but in a few cases (particularly those following skull injuries or nasal operations), it is necessary to consider ligation of a main artery of supply.

The purpose of this paper is to describe the blood-supply of the nasal mucous membrane in relation to the surgical control of severe epistaxis, and in addition to describe 7 cases of epistaxis in which the bleeding was not controlled until the blood-flow through the ethmoidal arteries on the affected side had been interrupted. It is not our purpose to discuss either the aetiology or local treatment of nasal hæmorrhage.

The arteries from which bleeding occurs in cases of epistaxis are not necessarily large vessels, but they lie in regions where they may easily suffer either mural injury or laceration. In addition, the tissue in which they are embedded is such that contraction and retraction of the vessels is relatively impeded, and thus the spontaneous arrest of hæmorrhage cannot so readily take place as it does elsewhere. Secondary hæmorrhage also may readily occur from these vessels in the presence of nasal sepsis.





Figs. 452, 453.—The arteries supplying the nasal mucous membrane. (*After Zuckerkandl.*)

THE BLOOD-SUPPLY OF THE NASAL MUCOUS MEMBRANE IN RELATION TO EPISTAXIS

The blood-supply of the nasal cavities was described in some detail by Zuckerkandl in 1885. His observations were based on the study of a number of specimens in which the blood-vessels had been injected. He states that the largest source of supply is from the sphenopalatine artery, but that the facial and the anterior and posterior ethmoidal arteries also contribute to the arterial network which supplies the interior of the nose. The sphenopalatine and facial arteries are derived from the external carotid artery, while the ethmoidal arteries are branches of the ophthalmic artery which springs from the internal carotid artery. The relative sizes, average positions, and areas covered by the arteries supplying the nasal mucous membrane, are given in *Figs.* 452, 453. These figures are based on the description by Zuckerkandl.

The branches from the sphenopalatine and facial arteries enter the nasal cavity through its lateral walls. Here, in the event of rupture or laceration, they are comparatively easy of access through the anterior nares, and hæmorrhage, although usually checked by packing or coagulation, may in certain instances be controlled under vision by ligation (Rosenvold, 1940). In some cases, owing to distortion of the nasal cavities occasioned by maldevelopment, injury, or disease, local measures may not be effective. In such an event, ligation of the external carotid artery on the affected side, in addition to local measures, has been considered the logical method of controlling the hæmorrhage. Johnson and Foster (1933) and Barker (1943) have reported instances in which such treatment was successful, and many other otolaryngologists will have had similar cases in their own experience. However, in some instances even ligation of the external carotid artery has failed to control the bleeding satisfactorily, and Hirsch (1936), among others, has advocated ligation of the internal maxillary artery in the sphenomaxillary fossa. Ligation in this position is said to circumvent the possibility of blood reaching the open vessel through anastomoses between the external carotid arteries across

the midline. The vessel is approached through the maxillary antrum, and the operation is described as being easy technically and can be performed quickly under local anæsthesia.

It is interesting, nevertheless, that severe bleeding from the nose which occasionally follows the operation of antrostomy, and which must involve branches of the sphenopalatine artery, is always successfully controlled by ligation of the external carotid artery.

The ethmoidal arteries pass into the nasal cavities through their roof. They are thus more difficult of access through the anterior nares, for not only are they some distance upwards and backwards from the nostrils, but the olfactory cleft is narrow in this position. Thus, in the event of their rupture

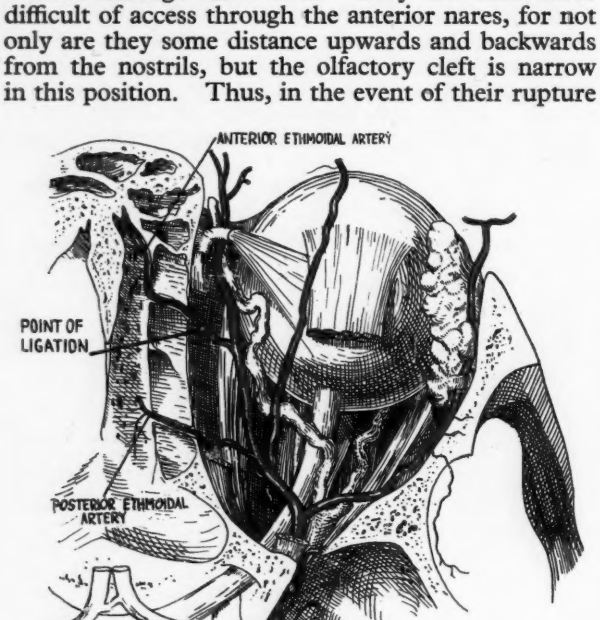


FIG. 454.—Showing the point of ligation of the anterior ethmoidal artery and the anatomy of the region. (*After Poirier.*)

or laceration (following injuries or operations in the region of the upper or middle concha), the control of hæmorrhage under vision or by packing is often difficult. In addition, in an injury which causes severe epistaxis, it is common for either the nasal or ethmoid bones to be fractured, a condition which tends to increase still further the inaccessibility of the ethmoidal arteries. Ligation of the external carotid artery, although diminishing the total blood-flow through the arterial network in the nasal mucous membrane, does not usually control the bleeding in such cases, and the next procedure advocated by most text-books is to tie the *common* carotid artery. It is true that except in very rare instances in which the ophthalmic artery springs from the external carotid artery (Adachi, 1938), the ethmoidal arteries derive their blood from the internal carotid artery. However, ligation of this vessel will not, in the majority of cases, control hæmorrhage from the ethmoidal arteries, owing to the anatomical efficiency of the circle of Willis. Adachi notes the absence of an anterior communicating artery only four times in 1653 dissections, and states that it may even be duplicated. In addition to anatomical evidence of the free anastomoses between the internal carotid arteries at the base of the brain, Schorstein (1940) has shown that the circle of Willis is very efficient from the clinical point of view. But, apart from the doubtful value of ligating the common carotid artery to control hæmorrhage from the ethmoidal arteries, such a procedure is not without danger, for Schorstein has also demonstrated that, although the collateral circulation is adequate under normal intracranial circulatory conditions, in the presence of anæmia due to hæmorrhage serious cerebral damage may ensue.

Clearly, the logical method of controlling hæmorrhage from the roof of the nasal cavities is by interrupting the flow of blood through the ethmoidal arteries themselves. They can be exposed in the orbit shortly after they spring from the nasal branch of the ophthalmic artery (*Figs.* 454, 455). The operation is an easy one and can be performed in a few minutes under local anæsthesia.

These anatomical facts do not appear to have been appreciated, for in no instance, either in current English or American text-books, is ligation of the ethmoidal arteries mentioned in connexion with the control of epistaxis, although ligation of the *common* carotid is recommended if ligation of the external carotid artery and local measures together are unsuccessful.

In the absence of any clear teaching on the subject of the surgical control of epistaxis, it has become traditional to ligate first the external, then the common, carotid artery in desperate cases. In a large number of instances this line of treatment is successful. In some cases this success must clearly have been because the correct artery was tied; in others, it is quite probably due to the combination of a *diminution* of the blood-flow following carotid ligation, local packing, and the administration of sedatives in sufficient doses to keep the patient quiet for a sufficient period after operation.

It is surprising that this state of affairs still exists, for ligation or coagulation of the ethmoidal arteries is a well-recognized step in the course of operative procedures in the neighbourhood of the ethmoidal air cells, and Davis (1939) describes a fatal case of epistaxis due to traumatic rupture of an anterior ethmoidal artery in which he says that life would have been saved had the artery been ligated in the orbit. Moreover, attention has been drawn to the possible value of ligation of the anterior ethmoidal artery in cases of intractable epistaxis by Goodyer (1937) and by Reading (1945), who each describe a single case in which a cure was effected by this simple procedure.

In rare instances severe epistaxis may result from rupture of an intracranial aneurysm into an accessory air sinus (Griffiths, 1915; Birley and Trotter, 1928; and Davis, 1939). Ligation of the common carotid artery on the affected side is the only treatment of any value in such cases. If there is any doubt as to the presence of an intracranial aneurysm, and the patient's condition allows of it, an arteriogram can always be performed before carotid artery ligation and the diagnosis established.

CASE REPORTS

In the case reports which follow, the primary causes of the epistaxis were injury (*Cases* 1–4);

hyperpiesis (*Cases* 5 and 7); and telangiectasis of pregnancy (*Case* 6).

Case 1.—Dvr. W. K., aged 23, was struck over the bridge of the nose by a heavy metal staple on Sept. 2, 1943. His nose bled so severely that the right nostril had to be packed at a Camp Reception Station. This appeared to be effective at the time, but next day there was a trickle of blood coming from the posterior nares. He was therefore sent to a Military Hospital for X-ray examination, and this showed a fracture of the nasal bones with dislocation to the left and backwards. The packing was changed and the bleeding ceased, but he was kept at the Camp Reception Station for six days as a precautionary measure.

On the sixth morning after the accident he was allowed up, and the nasal packing was removed. At 5 p.m. the bleeding recurred and the right nostril was repacked. This was only partially effective, for blood continued to trickle from the posterior nares.

Next day he was pale and restless. During examination, it was elicited that he was subject to headaches of migrainous type, and this raised the possibility that the nasal hæmorrhage was due to a ruptured intracranial aneurysm leaking into one of the nasal air sinuses. He was, therefore, transferred to a Military Hospital for Head Injuries.

On admission, examination of the nose revealed that blood was coming from above the middle concha on the right side, but it was not possible to localize its origin more accurately. The nostril was packed with gauze soaked in adrenaline and this appeared to control the bleeding; next morning, however, it recommenced. At this time the blood-count showed 1,500,000 R.B.C.'s and the hæmoglobin concentration was 38 per cent.

An arteriogram, rapidly effected under local anæsthesia, proved to be normal, but because carotid compression appeared to control the bleeding, the external carotid was tied. Bleeding, however, continued as before, and the patient was kept alive only by continuous and brisk blood transfusions. A discussion next arose as to whether the common carotid artery should be tied (in view of the cessation of bleeding on compression) or whether the simpler and less drastic operation of interrupting the ethmoidal vessels should be tried.

The latter operation was considered to be the most logical step and was therefore carried out. The anterior and posterior ethmoidal vessels were exposed under local anæsthesia, and coagulated with diathermy current. The bleeding ceased immediately and recovery was uninterrupted.

Comment.—In this case there was proof that the bleeding was coming from an ethmoidal artery. In the light of our subsequent experience in these cases, the site of origin of the bleeding, *above* the middle concha, would have led us to interrupt the ethmoidal vessels as the primary operative procedure. The collateral circulation through the circle of Willis was assumed to be poor in this case, for carotid compression controlled the bleeding. Presumably, therefore, serious cerebral damage might have followed ligation of the internal carotid artery.

Case 2.—J. W., aged 11, was hit on the nose by a 'bumping' cricket ball on May 27, 1944. Five days later he had a severe hæmorrhage from the left nostril. The bleeding was controlled by packing. The next day there was another severe hæmorrhage, also controlled by packing. He was admitted to hospital on June 3 for observation and had two further minor hæmorrhages that night. Examination of the septum revealed no definite bleeding vessel, but a suspicious vein was cauterized. A radiograph showed fractured nasal bones and a slight opacity of the sinuses on the left side. On June 8 he had a massive left-sided nasal hæmorrhage, which was controlled by packing. In view of the extent of the hæmorrhage, however, the left external carotid artery was ligated under cervical plexus block anæsthesia. Following this operation, the nasal pack was removed and the bleeding was found to have ceased. Compression of the common carotid artery was not carried out before the operation. On June 11 he had another severe hæmorrhage. This was controlled by means of packing in the anterior part of the roof of the nose. In view of this observation it was decided to ligate the anterior ethmoidal artery. This was carried out with stainless steel wire under general anæsthesia. Recovery was uninterrupted.

Comment.—The nature of the injury should have indicated that the source of the blood was most probably from the anterior ethmoidal artery, and indeed this was confirmed by the fact that a high anterior nasal pack controlled the hæmorrhage. This case illustrates the importance of noting the position within the nasal cavity in which packing has to be placed to control the bleeding. Even when the hæmorrhage is very profuse, it is possible by this means to locate the approximate site from which the bleeding is coming. The ligation of the external carotid artery must have reduced the blood-flow in the network of vessels supplying the nasal cavity and temporarily controlled the bleeding. It was, however, of no permanent value.

Case 3.—F/O. S., aged 24, was involved in an air crash on Aug. 25, 1944. He sustained a head injury, with severe epistaxis. The nasal hæmorrhage ceased spontaneously and he made good progress until September 5, 1944, when he had a further profuse hæmorrhage from the left nostril. On this occasion, it was estimated that he lost between two and three pints of blood. He was transfused, the nose was packed tightly, and the bleeding ceased. On the next day there was a further severe hæmorrhage, but on this occasion it could not be controlled completely by packing. The patient was therefore transfused again and then anæsthetized and the packing removed. Blood was seen to be coming from high up in the region of the olfactory cleft. The nose was repacked, and he was transferred to a Military Hospital for Head Injuries.

On admission he was pale and restless. Radiographs of the skull revealed no fracture and there was no clinical evidence of an intracranial aneurysm. Removal of the nasal packing was followed by severe arterial bleeding, and his condition became critical. He was given a further blood transfusion and the carotid arteries were exposed in the neck. The external carotid artery was ligated, but immediately afterwards the patient expelled his nasal packing during a fit of coughing and the bleeding again became violent. The internal and common carotid arteries on the left side were then tied, but without appreciably diminishing the loss of blood from the nose. Once again, the nostril was packed and he was given a third blood transfusion. No abnormal neurological symptoms or signs followed these procedures.

Next day the nasal packing became loose, he began to bleed again and was only kept alive by continuous brisk blood transfusion. By the following evening he was desperately ill, for by now he was losing blood faster than he was receiving it. It was therefore decided to try the effect of occluding the ethmoidal vessels. This was done under regional block anæsthesia of the first and second divisions of the fifth cranial nerve. The anterior ethmoidal artery and an accessory vessel were occluded. The nasal packing was removed and it was found that the bleeding had ceased. The patient made an uninterrupted recovery.

Comment.—The source of the bleeding from high up in the olfactory cleft should have suggested that the ethmoidal vessels were implicated. The efficiency of the circle of Willis was shown by the fact that on ligation of the internal carotid artery the bleeding was unaffected; and, although the patient was exsanguinated, he suffered

no neurological complications following ligation of the common carotid artery.

Case 4.—Sgt. J. H., aged 34, had a severe spontaneous left-sided epistaxis on Nov. 23, 1944. His medical officer packed his nose but this did not control the bleeding, so he was transferred to hospital. The packing was removed and this was followed by brisk arterial hæmorrhage. The bleeding came from high up in the nose, but, as the septum was deviated to the left, the bleeding point could not be located precisely. The nose was, therefore, repacked. The next day blood was still trickling from the nose, and removal of the packing was accompanied by a brisk hæmorrhage. The subsequent pack was left undisturbed for two days; nevertheless, slight bleeding continued. On the third day a submucous resection of the septum was carried out in order to localize the bleeding point with greater accuracy. However, it was only possible to confirm that the bleeding was coming from high up in the olfactory cleft. Following the operation the nose was repacked tightly and the patient given a blood transfusion.

Three days later, despite packing, the bleeding again became profuse. The common carotid artery was, therefore, ligated under general anæsthesia. In spite of this, brisk arterial bleeding recommenced from the left nostril when the nasal packing was removed. No abnormal neurological symptoms or signs followed the occlusion of the common carotid artery.

Two days later the patient's condition had become critical, and it was decided to occlude the ethmoidal vessels. The anterior ethmoidal artery was ligated and the posterior coagulated with a diathermy current. When the nasal pack was removed there was no further bleeding and from then onwards the patient's recovery was uneventful.

Comment.—There was good evidence in this case also that the bleeding was coming from the anterior ethmoidal artery. Unfortunately, compression of the common carotid artery was not carried out before it was ligated. It is of interest that ligation of this artery was not followed by damage to the central nervous system. *Cases* 3 and 4 lend support to Schorstein's contention that the collateral circulation through the circle of Willis is usually sufficient to prevent cerebral damage following ligation of a common carotid artery.

Case 5.—J. B., aged 12, was struck on the nose by the leg of a chair early in July, 1945, but there was very little bleeding from the nostrils. Four weeks later, however, his left antrum was punctured and washed out because it was infected. On the evening of Aug. 14 he was struck a trivial blow on the nose by his sister. A severe epistaxis from the left nostril followed immediately. Medical attention was not sought, for he was camping at the time and no doctor was readily available. First-aid treatment was given, but he apparently lost a considerable quantity of blood during the night. The next day he was admitted to hospital and the first-aid nasal pack was removed. The hæmorrhage was minimal by now, but, nevertheless, an enlarged vein in Little's area was cauterized as a precautionary measure. A severe deviation of the septum to the left was noted. A radiograph of his sinuses showed a fracture into the left frontal sinus, and possibly into the left ethmoidal region, and there was relative opacity of both the left antrum and left ethmoidal region.

Despite the cauterization, slight bleeding recommenced and on Aug. 17 it had increased and become bright red. The left anterior ethmoidal artery, therefore, was exposed and occluded under local anæsthesia. The fracture into the frontal sinus involving the vessel was identified during the operation. There was no further bleeding.

Comment.—In this case the anterior ethmoidal artery was occluded as a primary operative procedure and was effective. The decision to occlude the artery was based on the position from which the hæmorrhage was proceeding. The sequence of pathological events probably took the following form: fracture through the foramen for the anterior ethmoidal artery, infection within the left sinuses, erosion of the vessel wall, initiation of epistaxis by a trivial injury.

Case 6.—Mrs. C. I., aged 30, during the evening of Aug. 16, 1945, had a severe hæmorrhage from the right nostril; this was controlled by means of a nasal pack. Twenty-four hours later when the packing was removed, the bleeding recommenced. Another pack was inserted and she was sent into hospital next day, for she had lost a considerable amount of blood. There was no history either of injury or infection. She was six months pregnant: it was her second pregnancy and there had been no epistaxis on the previous occasion. There was a trace of albumin in her urine, but her blood-pressure was not raised, she had no œdema of the ankles or face, and her blood-urea level was within normal limits.

Shortly after her admission, the pack was removed and the bleeding recommenced. The blood was seen to be coming from the roof of the right nasal cavity, anteriorly between the septum and the middle concha. The precise origin of the bleeding could not be determined, however, owing to a deviation of the septum towards the right. The nasal mucous membrane was covered with a paste containing 25 per cent cocaine and desiccated suprarenal gland, and a cotton-wool pack was inserted into the nostril. Bleeding ceased in a few minutes and the septum was then cauterized as high up as possible above the middle concha.

A small quantity of blood continued to pass down the pharynx for the next four days until, on Aug. 22, another massive hæmorrhage occurred. By this time her erythrocyte count had dropped to 1,800,000 and she was given a transfusion of two pints of blood. The bleeding was again controlled by packing, but, in view of her condition, it was decided to occlude the right anterior ethmoidal artery. This was carried out under local infiltration anæsthesia, and when the nasal packing was removed the bleeding did not recur.

Comment.—In this case also the anterior ethmoidal artery was occluded as the primary surgical procedure and effected a cure. Telangiectasis of pregnancy is a recognized disease, and in the absence of injury or sepsis it is presumed that this patient had such a condition high up in the right olfactory cleft.

Case 7.—G. B-T., aged 62, on Jan. 12, 1946, had a sudden profuse epistaxis from the left nostril while repairing a wheel of his car. He lay down and it stopped after there had been a considerable loss of blood. Next day he had another large hæmorrhage, which was controlled by packing. The following night there was some oozing, and when the packing was removed the bleeding again became profuse. This was controlled by further packing, but he vomited on his way to hospital and the bleeding recurred.

On arrival at hospital the bleeding again became profuse when the pack was removed. The source of the bleeding was anterior and superior to the middle concha. The bleeding was controlled by a pack of cotton-wool covered with 25 per cent cocaine paste inserted into this region.

The patient's general condition was fair, but the percentage of hæmoglobin had fallen to 38. Accordingly, the anterior ethmoidal artery was interrupted with two silver clips under local infiltration anæsthesia. The vessel, it was noted, was larger than usual.

The following morning the patient had another considerable hæmorrhage, which came from the region supplied by the anterior ethmoidal artery. The wound was reopened; the vessel was ligated with a stainless

steel wire and coagulated with a diathermy current through the wire. In addition, the posterior ethmoidal artery, which was very small, was also coagulated. There was no further bleeding.

Comment.—In this case the bleeding was associated with hyperpiesis, and was arising from the area supplied by the anterior ethmoidal artery. Packing only temporarily controlled the bleeding. The failure of the first operation was due either to the fact that the anterior ethmoidal artery was duplicated, and the silver clips not applied to both vessels, or to the fact that the posterior ethmoidal artery was responsible for the hæmorrhage. In our opinion, the former is more likely, for the posterior vessel was so small. Complete occlusion of the vessels, as in our previous cases, stopped the bleeding.

OPERATIVE TECHNIQUE

Anæsthesia.—Local infiltration of procaine or nupercaine and adrenaline is the method of choice, for it is quick and less complicated than regional block anæsthesia. General anæsthesia is contraindicated because of the danger of aspiration of blood into the lungs.

The skin over the bridge and side of the nose on the affected side is infiltrated, first intradermally, and then subperiosteally, with a 1-in. 21-gauge needle. A 2-in. needle of the same gauge is then substituted and the medial wall of the orbit infiltrated subperiosteally. It is important that the needle point should be in contact with the bone throughout, to avoid injecting fluid into the orbit itself, and it should be directed so as to infiltrate above and below the point of entry of the ethmoid vessels into the nasal cavity.

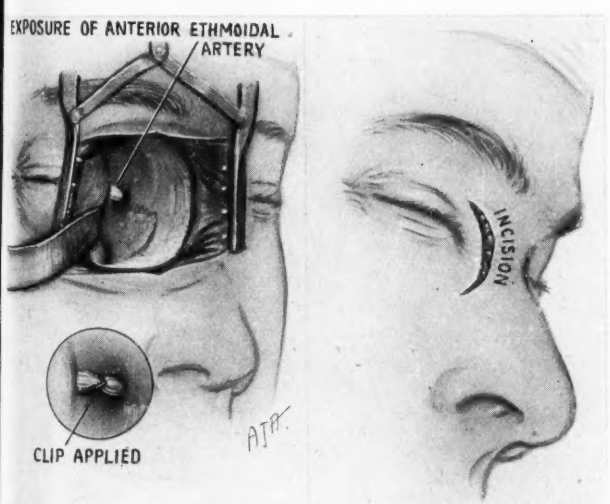


FIG. 455.—Exposure of the anterior ethmoidal artery.

Operation.—A curved 1½-in. incision, with its centre situated medial to the inner canthus of the eye, is employed, and is carried to bone. The periosteum is incised and elevated from above downwards and backwards to a depth of about 1½ in. from the bridge of the nose, when the ethmoidal vessels will be seen passing in a cuff of connective tissue to the foramen in the suture line between the frontal and ethmoid bones along the inner wall of the orbit. The vessels are easily defined (*Fig.* 455), after which a malleable metal retractor is placed beneath the periosteum and the orbital contents retracted gently outwards and downwards. Ligation is relatively difficult in this situation, so that a silver clip has usually been employed to interrupt the anterior ethmoidal artery. The posterior ethmoidal vessel or vessels are usually small and can be coagulated with a high-frequency diathermy current. This manœuvre requires care, for the optic nerve is only a very short distance behind the posterior ethmoidal arterial foramina. If the skin edges are carefully approximated the resulting scar is practically invisible.

DISCUSSION

Epistaxis may result from the rupture or laceration of branches of the external carotid artery or of the ethmoidal arteries. This statement is supported by anatomical and clinical considerations. If the hæmorrhage originates from an ethmoidal artery, we have demonstrated that it cannot be effectively controlled by ligating either the common, the internal, or the external carotid artery.

It has been recommended by Hirsch (1936) that the internal maxillary artery should be ligated in preference to the external carotid artery, for he has stated that failure to control epistaxis by carotid ligation is due to anastomoses between the external carotid arteries across the midline. On the other hand, our experience, particularly in the cases reported, suggests that failure of carotid ligation is due to direct leakage from the ethmoidal arteries. Furthermore, it is our belief that ligation of the internal maxillary artery is a difficult, dangerous, and unnecessary procedure. We believe that ligation of the common carotid artery is never justifiable for the control of epistaxis, unless this is due to a ruptured intracranial aneurysm leaking into an accessory air sinus.

We recommend that in cases of severe epistaxis diagnosis and treatment should proceed along the following lines: It must first be determined whether the bleeding is coming from above or below the middle concha. In cases where the hæmorrhage is profuse, the approximate source can be determined by observing the effect of packing different regions of the nasal cavity. Digital pressure on the common carotid artery may be used as an additional aid. If the compression does not arrest or conspicuously diminish the hæmorrhage, this would strongly suggest that it originates from an ethmoidal artery.

The approximate source of the bleeding having been determined, local measures such as packing should be instituted. If local measures fail, then either the external carotid or ethmoidal arteries should be occluded under local anæsthesia, depending on the source of the hæmorrhage.

SUMMARY

1. The blood-supply of the nasal mucous membrane has been discussed in relation to the surgical control of epistaxis.

2. Seven cases of epistaxis have been reported in which the hæmorrhage was not controlled until the ethmoidal arteries on the affected side had been occluded. In three of the cases reported the ethmoidal arteries were occluded as the primary and only surgical procedure.

3. The anatomical approach to the surgical treatment of epistaxis has been discussed.

The authors' thanks are due to Mrs. M. Clark for preparing the illustrations for *Figs.* 452–454 and to Miss A. Arnott for the drawing of *Fig.* 455.

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SHORT NOTES OF RARE OR OBSCURE CASES

RETROGRADE INTUSSUSCEPTION OF LESSER CURVATURE OF STOMACH, PYLORUS, AND FIRST PART OF THE DUODENUM INTO THE ŒSOPHAGUS

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APART from pyloric stenosis, surgical conditions of the stomach are rarely found in infants. This case is an example of a retrograde intussusception into the lower œsophagus of the lesser curvature, pylorus, and the first part of the duodenum. It is recorded because of the unusual nature of the findings and the absence of any reference to a similar condition in the available literature.

CASE REPORT

This 10-months-old child had been admitted to hospital because of malnutrition. The records reveal that the child was markedly dehydrated, had a temperature of 103° on admission, and despite feedings went rapidly downhill until death five days after admission. X rays revealed a markedly dilated, globular stomach. No further clinical information was available.

Post-mortem examination (*Figs.* 456–458) showed that the stomach (S) was globular in shape and about 6·5 cm. in diameter. The pylorus and first part of the duodenum were not visible anteriorly. Extending from the right

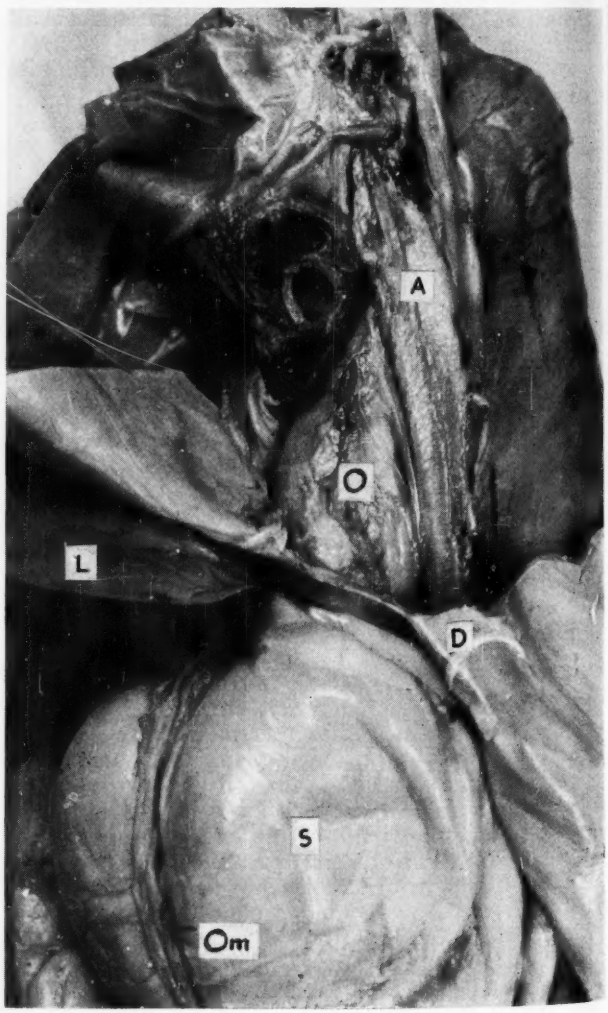

FIG. 456.—Showing dilatation of œsophagus in relation to diaphragm and aorta. A, Thoracic aorta; D, Diaphragm; O, Œsophagus; L, Liver; Om, Attachment of omentum; S, Stomach.

of the œsophageal opening above, to the spleen on the left below, an oblique attachment to the stomach of the greater omentum was observed (Om). There was no connexion between this membrane and the transverse colon, except at its splenic extremity.

From the transverse colon, the mesocolon, triangular in shape, extended and was attached by its apex to the first part of the duodenum. The latter could be seen

as a thickened, narrow, cord-like structure extending towards the diaphragm, and was lost to view in the region of the œsophageal hiatus.

Above the diaphragm the distal end of the œsophagus (O) was dilated for a distance of 5 cm. Its widest diameter measured 2·5 cm. Within it could be palpated into the œsophagus for a distance of 5 cm. beyond the level of the diaphragm, taking with it the pyloric antrum, the pylorus, and proximal portion of the duodenum. These formed a compact cone which had caused almost complete œsophageal obstruction (C). However, a probe could easily be passed alongside the right border

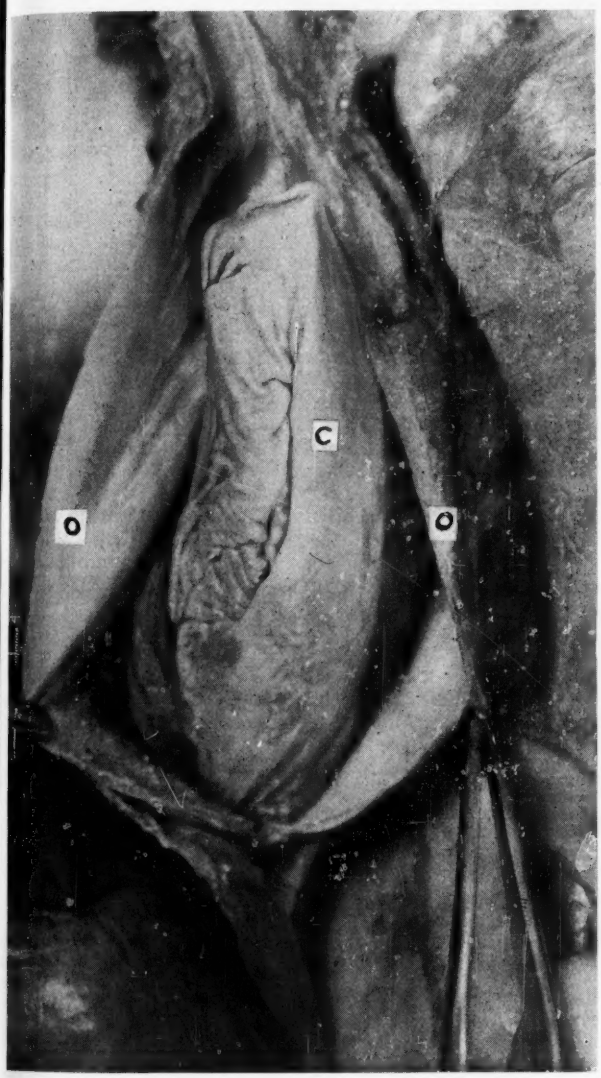

FIG. 457.—Showing O of *Fig.* 456 with a vertical incision to expose the contents. C, Cone of lesser curvature, pylorus, and duodenum; O, Edges of incised œsophagus.

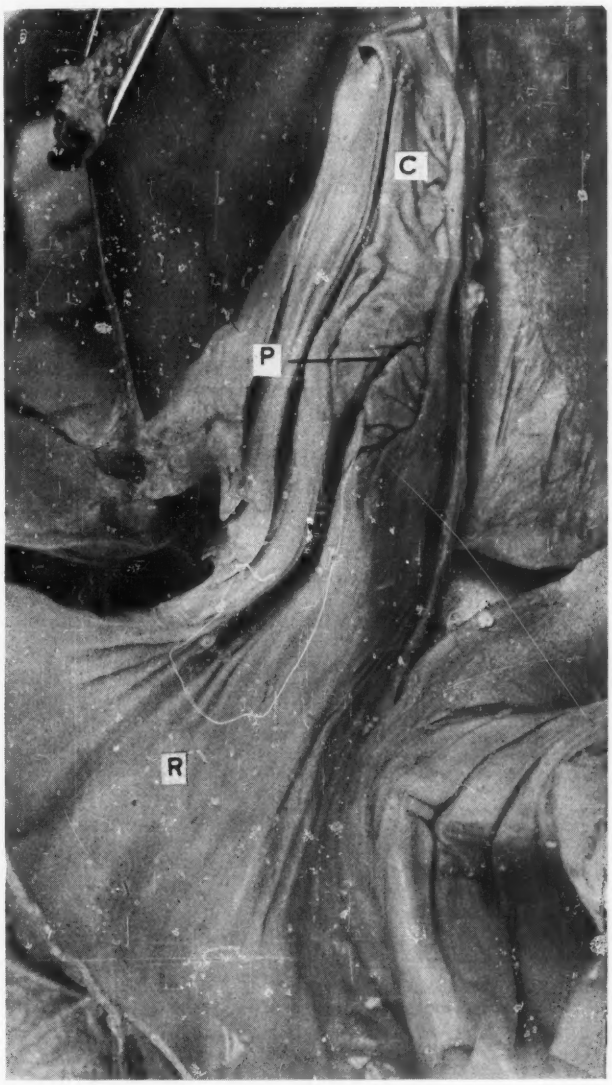

FIG. 458.—The incision through the œsophagus has been extended into the anterior wall of the stomach to expose its interior. C, Cone of lesser curvature as before; P, Opening of pylorus; R, Mucosa of stomach.

what eventually was shown to be the lesser curvature, pylorus, and proximal part of the duodenum (*Fig.* 457).

An incision opening the anterior wall of the stomach and extending to the upper limit of the œsophageal dilatation showed the following features (*Fig.* 458). The contents of the stomach consisted of an homogeneous, cheese-like mass. The wall of the stomach was parchment-thin and all its rugosities had completely disappeared (R). The lesser curvature had invaginated of this mass into the stomach. The opening of the pylorus was not visible until the longitudinal rugæ of the lesser curvature within this mass were separated. The pylorus was therefore situated in the œsophagus (P).

The apex of the transverse mesocolon, to which reference has already been made, was attached to the antero-superior surface of the proximal 2 cm. of the duodenum. The upper part of the transverse mesocolon was contained within the œsophagus.

A CASE OF BILATERAL PNEUMOTHORAX AND BILATERAL PENETRATING WOUNDS OF THE DIAPHRAGM WITH RECOVERY

By A. J. P. GRAHAM

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PENETRATING wounds of the chest wall may result in injuries to abdominal organs, and the following is a description of a case in which bilateral injuries of the chest wall resulted in bilateral pneumothorax, penetration of both sides of the diaphragm with rupture of the spleen and liver, with complete recovery.

CASE REPORT

E. B., aged 28 years, a corporal in the A.T.S., was admitted after a road accident. Sergeant B. L. was admitted at the same time with almost exactly similar injuries to the chest wall. As they had been travelling in the same lorry it appeared, therefore, that the mechanism of their production must have been similar. Another vehicle had collided with the side of the lorry in which they had been travelling. Both had been sitting against a wooden slat running along the inside of that side of the lorry struck by the other vehicle. The force of the impact had evidently caused the wood against which they were leaning to give way behind them, and the two broken ends of the slat had penetrated each side of the chest wall in both cases.

In the second woman splinters of wood were later removed from the tissues in front of both scapulæ several inches away from the entry wounds in the mid-axillary regions.

On admission E. B. was in considerable pain, which was most severe in the lower thoracic region at the back. On immediate examination she was found to be suffering from shock with a rapid feeble pulse, pallor, and obvious dyspnœa. There was no evidence of severe blood-loss externally apart from blood-stained uniform and bandages.

Measures for the treatment of shock were employed, and half an hour after admission she had recovered sufficiently to allow further examination to take place. The blood-pressure was 125/85, pulse-rate 92, respirations 30 and shallow. There was no obvious cyanosis. The apex beat was displaced to the left, the exact distance not being measured. On removing the first-aid dressing two thoracic wounds were found. That on the left was a penetrating wound through the eighth and ninth intercostal spaces and eighth and ninth ribs in the anterior axillary line. Protruding from it were part of the omentum and some bowel. On the right an almost similar wound through the eighth and ninth spaces and ninth rib in the posterior axillary line was found. Through this air passed in and out with every respiration. This wound was promptly covered with a sterile dressing. There was no marked difference between the percussion note on the two sides, though on the right side air entry was faint. The abdomen was rigid, especially in the upper quadrants. No other serious injuries were found.

The patient's general condition improved rapidly, and an hour and a half after admission her blood-pressure was 150/100, pulse 100.

X-ray Examination revealed fractures of the ninth and tenth ribs behind the angles and in the posterior axillary line, and single fractures of the eleventh and twelfth ribs in the posterior axillary line on the right side of the chest. On the left there were fractures of the eighth and ninth ribs in the mid-axillary line. Examination of the lung fields showed a right pneumothorax with the heart shifted to the left, a partial pneumothorax and generally increased density of the lower half of the lung on the left. The latter did not appear to correspond strictly to a lower-lobe collapse, part of the upper lobe being also involved.

Operation was decided on for the purpose of closing the chest wall and returning the omentum and bowel to the abdomen.

AT OPERATION.—A transfusion was set up and two pints of Group O blood were given during and after operation. The prolapsed omentum was ligated and excised. The bowel, part of the transverse colon, was undamaged and was returned to the peritoneal cavity through the tear in the diaphragm. It was then seen that the left lower lobe was retracted two or three inches above the diaphragm, the space being occupied by air and communicating freely with the atmosphere through the chest wall, and with the peritoneal cavity. Protruding through the hole in the diaphragm was the upper pole of a ruptured spleen and a small accessory spleen the size of a grape. It was obvious that there was at least a partial pneumothorax on both sides. The condition of the patient was surprisingly good, and at no time did her respiration or heart give the anæsthetist much anxiety. A close-fitting mask with a Boyle's machine maintained a certain degree of positive pressure, giving gas, oxygen, and ether.

As it was necessary to excise the spleen, the original chest wound was continued downwards across the costal cartilages and then transversely across the abdomen. A considerable quantity of blood was found in the peritoneum, and bleeding was continuing from the splenic pedicle. Splenectomy was performed by Mr. E. W. Sheaf. A rapid survey of other abdominal organs was made. No intestinal perforations were found, but palpation of the under surface of the diaphragm on the right revealed another tear through which the sharp ends of fractured ribs protruded. On examining the upper and posterolateral surfaces of the liver several lacerations were felt, the largest appearing to be about a quarter of and inch deep and one and a half inches long. These had been caused by the impaction of fractured ribs. It was considered unnecessary to suture the liver. After the abdominal incision was closed the tear in the left side of the diaphragm was sutured with catgut. Blood-clot was cleared from the left pleural cavity and the pleura and muscle layers resutured without drainage. Vaseline gauze and sulphanilamide powder were applied and the skin wound left open at the site of the original chest-wall injury.

Attention was then directed to the wound on the right. The dressing was removed from the opening. Free entry and exit of air now occurred with every respiratory movement, but as far as could be ascertained there was no mediastinal flutter and no marked cardiac or respiratory embarrassment. A rapid wound excision with removal of sharp rib ends and loose bone fragments was carried out. The diaphragm and chest wall were closed without drainage as on the opposite side. According to d'Abreu routine closure of the chest without drainage is preferable to tube drainage provided post-operative collections of fluid in the pleural cavity are aspirated thoroughly. In this case it proved successful.

PROGRESS.—Post-operatively continuous oxygen was administered through a B.L.B. mask, and twelve hours later sulphathiazole in full doses and systemic penicillin, 50,000 units first dose and 30,000 units 3-hourly, were commenced. This dose of penicillin was maintained for

seven days, then half the dose was continued 3-hourly for a further two days. When the temperature had settled to normal for two days on completion of this course a total of 1,415,000 units had been administered. The highest recorded temperature was 101·2° twenty-four hours after operation. Two days after the injury the respirations were forty per minute, but by four days they had returned to normal.

Chest examination twelve hours after operation showed faint air entry on the right with a little fluid in the pleural cavity. On the left the air entry was much better, though breath-sounds were harsh in character and accompanied by a few rales. Coughing was encouraged by the use of potassium iodide, with small doses of morphine to depress pain sensation but not the cough reflex. This appeared to be quite successful, as sputum was easily cleared. Deep breathing exercises with carbon dioxide and oxygen for five minutes hourly were begun on the first day and lung expansion exercises performed twice daily with a masseuse.

Four days after operation the heart was still displaced to the left, though less than formerly. There was generally increased density of the left lower lobe. On the right there was no fluid and air absorption was complete except in the apical region. Respirations were normal at rest, but dyspnœa was evident on exertion. This gradually passed off as the lungs expanded.

Recovery was uneventful. A chest radiograph five weeks after operation showed no abnormality except for non-union of the eleventh rib. This caused a slight clicking, but no pain.

Discussion.—A case of bilateral abdomino-thoracic injury is described. It is of unusual interest in that one complete lung and part of the other were collapsed. In spite of this, the vital capacity was not reduced beyond that compatible with life and with treatment the patient made a complete recovery.

My thanks are due to Mr. E. W. Sheaf for permission to publish this case and for his advice throughout.

A CASE OF FILARIAL FUNICULITIS

By Surgeon Lieutenant Commander H. R. I. WOLFE, R.N.V.R.
and Surgeon Lieutenant Commander A. L. SCHOFIELD, R.N.V.R.

In these days of global warfare and consequent movements of Service personnel, tropical diseases and their sequelæ are much less confined to the tropics. The following case of filarial funiculitis, occurring nine months after the patient's return from Africa, provides an example of the importance of being alive to the possibility that the symptoms complained of may be a manifestation of a previous symptomless tropical infestation.

CASE REPORT

History.—The patient, G. A., a Naval Air Gunner, aged 25. At 17.30 hr. on Feb. 9, 1945, he felt a stab of pain in the left groin while climbing down from an aircraft to the flight deck. He attributed this to the pressure of his parachute harness. Later there was slight pain and swelling of the left scrotum, but he slept well. Next morning, on rising, the pain and swelling were still present and became worse as he went about his duties. He reported at the Sick Bay.

Previous History.—Measles aged 6, diphtheria aged 10, and in 1939 pneumonia. In March, 1942, he was drafted to West Africa, where he spent four weeks in the jungle. In June, 1942, he was transferred to East Africa, being stationed in a game reserve, where he remained until April, 1944. In June, 1943, a small right hydrocele, which had been present for five years, suddenly increased in size. A radical cure was performed, with an uneventful convalescence, and there has been no recurrence. On his way home in May, 1944, he developed his first attack of malaria when three days out from Mombassa. Apart from this illness and the operation for hydrocele, he was perfectly fit during the whole commission.

On Admission.—At 20.15 hr. on Feb. 10, when his ship returned to her base, he was discharged to this hospital as a case of suspected strangulated inguinal hernia. He was lying confortably in bed without rise in either temperature or pulse-rate (T. 97·6°; P. 76). The scrotal skin on the left side was indurated and œdematous, but the left testicle and epididymis, surrounded by a small lax hydrocele, felt normal. Extending from just above the left globus major to the external abdominal ring, there was a fusiform, tender thickening of the cord. This was less marked in the inguinal canal.

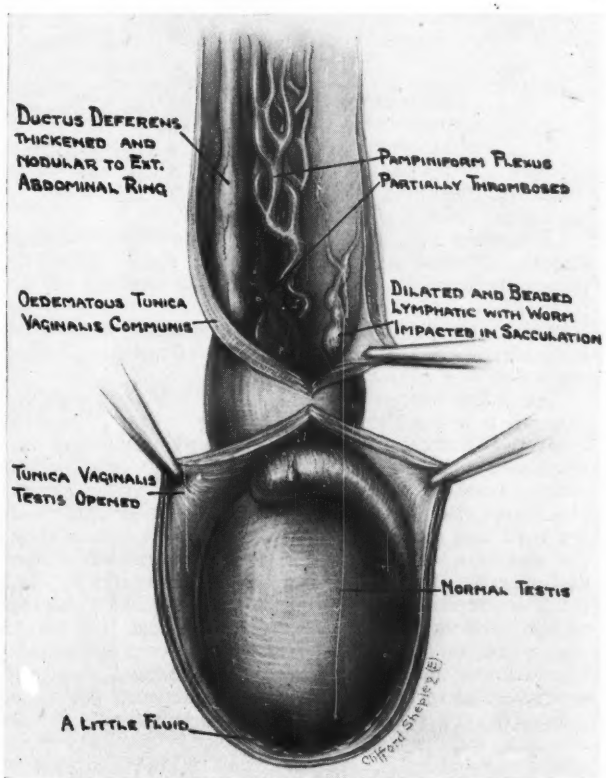
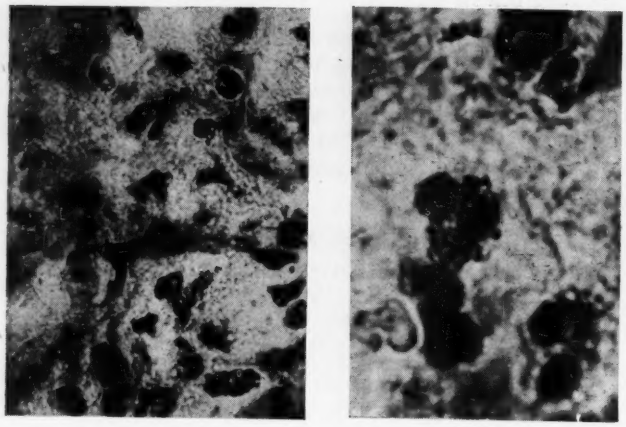

Ductus Deferens thickened and nodular to Ext. Abdominal Ring
Pampiniform Plexus Partially Thrombosed
Oedematous Tunica Vaginalis Communis
Dilated and Beaded Lymphatic with Worm Impacted in Sacculation
Tunica Vaginalis Testis Opened
Normal Testis
A little Fluid
Clifford Shepiro (E)

Fig. 459.—Drawing of findings on exploring cord and testicle.

Tenderness was elicited in the left iliac fossa and hypogastrium. Apart from slight thirst, no other abnormal signs or symptoms were noticed. No rigor, headache, malaise, anorexia, nausea, or vomiting. The bowels were regular and micturition was normal. Examination of the rectum, chest, and cardiovascular system revealed no other abnormal physical signs.



A B

FIG. 460.—Microphotographs of sections of nodules taken from surface of vas deferens. A, × 600; B, × 1500.

Blood-count :—

R.B.C.	5,000,000	
Hb	98	per cent
C.I.	1	
W.B.C.	7,400	
Polymorphs	62	per cent
Lymphocytes	23	,, ,,
Monocytes	9	,, ,,
Eosinophils	4	,, ,,
Basophils	2	,, ,,

Urine: Nothing abnormal detected.

Following rest in bed, the scrotal swelling subsided almost completely, but the tender mass above the testicle remained. On getting up, the swelling quickly recurred.

On a pre-operative diagnosis of torsion of a testicular hydatid, or an acute funiculitis of unknown aetiology, operation was advised.

AT OPERATION.—The cord and testicle were explored through a left inguinal incision (*Fig.* 459). The cord from the internal to the external abdominal ring was thickened and œdematous, and a watery exudate was visible beneath its coverings. Between the external abdominal ring and the globus major of the epididymis the cord had expanded into a firm fusiform mass about the size of a walnut. The tunica vaginalis was incised and found to contain a little clear watery fluid. The testis and epididymis appeared normal. The coverings of the cord were incised from the internal ring to the upper pole of the testis. The former were about $\frac{1}{8}$ in. in thickness below the external abdominal ring and much less so in the inguinal canal. Between the upper pole of the testis and the external abdominal ring the vas deferens was inflamed and nodular. On its surface was a number of discrete yellowish nodules varying in size from 0·5 mm. to 3·0 mm. in diameter. Some of these were sent for microscopy (*Fig.* 460). The pampiniform plexus was engorged and some of the radicles in contact with the inflamed vas were thrombosed. On dissection, an enormously dilated and varicose lymphatic was found running in the posterior coverings of the cord down to the upper pole of the testis. Through its thin wall the wriggling, white, hair-like coils of a worm were seen. The lymphatic, with the worm inside, was excised and the specimen sent for examination (*Fig.* 461). A blood-smear was taken and the wound then closed in layers.

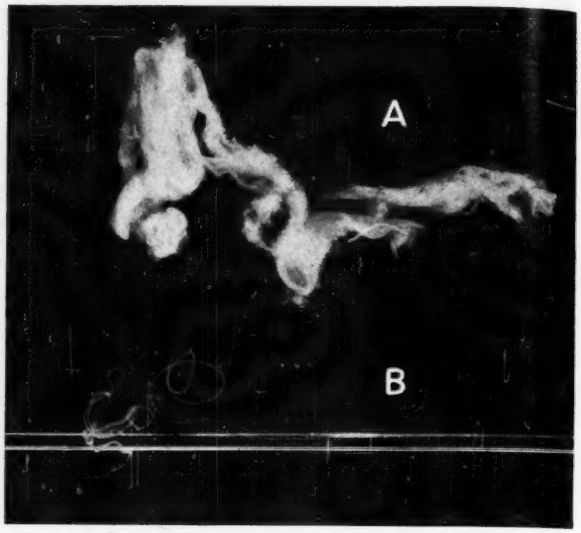


FIG. 461.—A, Lymphatic on 00 catgut; B, Worm on finely drawn capillary tubing. (× 3.)

PROGRESS.—Convalescence was uneventful, with healing by first intention. The scrotal œdema subsided in a week, and the funiculitis had almost completely resolved on his discharge from hospital three weeks later. Two months after operation there were no abnormal signs or symptoms.

PATHOLOGICAL REPORTS.—Daily blood examinations at 22.30 hr. for three weeks, and that of the specimen taken during the operation, failed to show microfilaria. Further diurnal blood examinations at 22.30 hr. and 10.30 hr. for a period of a week, one month later, were also negative.

The worms were identified as *Filaria bancrofti* by Lieutenant-Colonel W. F. Harvey, I.M.S. (Ret.).

The nodules taken from the surface of the vas deferens were found to consist of lymphoid follicles, packed with eosinophils, and with marked lymphangiectasis (*Fig.* 460).

COMMENT

Though cases of filarial funiculitis must be rather unusual in this country, Lt.-Colonel Grieg states that it is reported to be very prevalent among personnel serving in the Pacific theatre of operations (Fogel and Huntington, 1944). With the return of Service men from Africa and the Pacific it is not unlikely that further cases will be seen, and therefore when a case of funiculitis of obscure aetiology is encountered, the possibility of filarial infestation should be borne in mind.

We wish to thank Surgeon Captain H. F. Briggs, R.N., for permission to publish this case. We express our thanks to Lt.-Col. W. F. Harvey, C.I.E., I.M.S. (Ret.) and Lt.-Col. E. D. W. Grieg, C.I.E., I.M.S. (Ret.) for their help and advice; to Mr. Pettigrew for the photographs; and to Mr. Clifford E. Shipley for the drawing.

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BAYONET WOUND OF THE TRANSVERSE COLON

By Surgeon Commander A. LONG, R.N.

CASE REPORT

A cadet, aged 18 years, while undergoing training at a Pre-O.C.T.U. Centre, jumped from a height of nine feet on to the fixed bayonet of the man immediately in front. This entered his abdomen and there was some brisk hæmorrhage from the wound. On being seen soon afterwards by a Medical Officer, he was found to be moderately shocked and two pints of reconstituted serum were given. He was admitted to R.N. Sick Quarters one hour afterwards.

On Admission.—Temperature, 97°; pulse, 88; blood-pressure, 108 mm. systolic; pallid and rather shocked. Abdomen—small incised wound of entry of bayonet just external to the right rectus muscle on a line between the anterior superior spine and the umbilicus and the wound of exit of the point below twelfth rib 2 in. from midline in the costovertebral angle. Some brisk hæmorrhage from wound. Generalized tenderness and rigidity. An intravenous drip of glucose and saline was commenced and two pints of serum given in the course of the next hour.

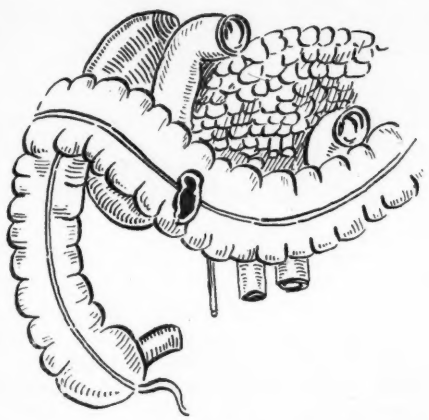


Fig. 462.—Drawing showing perforating wound of transverse colon.

At Operation (one hour later).—Under nitrous-oxide, oxygen, and ether anæsthesia, the abdomen was opened in the midline at the level of the umbilicus. Fæcal smell and gas were present on opening the peritoneum. Small amount of blood-clot evacuated from the right iliac fossa. Two gaping wounds of the transverse colon were found in the region of the hepatic flexure. These were immediately firmly sutured. Small intestine and other viscera were examined and found intact. During this time blood was seen entering the peritoneal cavity from the wound in the right iliac fossa, and on exploration it was found that the deep epigastric artery was severed. Hæmostasis was secured and a large drainage tube inserted through this wound after excision of skin edges; 5 g. sulphonamide powder was sprinkled over the suture line of the colon. Wound of exit treated similarly. Closure.

Progress.—Immediately following operation the patient was placed in a high Fowler's position. One gramme soluble M & B 693 was given into the intravenous drip and repeated at three-hourly intervals until consciousness was regained; then 1 g. was given four-hourly until a total of 31 g. had been given. The glucose-saline drip was kept in situ for five days, giving approximately four to five pints in twenty-four hours, and fluids were restricted by mouth. For the first three days he complained of severe colicky pains in the abdomen and morphine was exhibited freely. The tube drained well and was removed on the fourth day. Some slight elevation of temperature occurred during the first three days, afterwards becoming apyrexial; he vomited on one occasion only. Bowels were opened normally on the fifth day and afterwards convalescence was uneventful. Sutures were removed on the ninth day. All wounds healed well; slight hyperæsthesia was present over the front of the right thigh (L. 2 and L.3). The patient was free from symptoms and was discharged to convalescence soon afterwards.

Summary.—

1. Penetrating wound of abdomen, a bayonet passing completely through the peritoneal cavity and emerging below the right twelfth rib, inflicting damage only to the transverse colon.
2. Immediate operation and wounds of colon sutured.
3. Free drainage, high Fowler's position, restriction of fluids by mouth, and continuous intravenous drip of saline and glucose for five days following operation.
4. Chemotherapy: 31 g. of M & B 693 given in five days.
5. Uneventful recovery.

A CASE OF MULTIPLE DUCT PAPILLOMA OF THE MALE BREAST

By JAMES MORONEY

VISITING ASSISTANT SURGEON, BROADGREEN HOSPITAL, LIVERPOOL

The occurrence of a duct papilloma in the male breast is most unusual.

CASE REPORT

History.—A man, aged 31, was admitted to hospital complaining of an intermittent blood-stained discharge from the right nipple for two years and the presence of a swelling for two months. His father had had an operation on his breast many years ago.

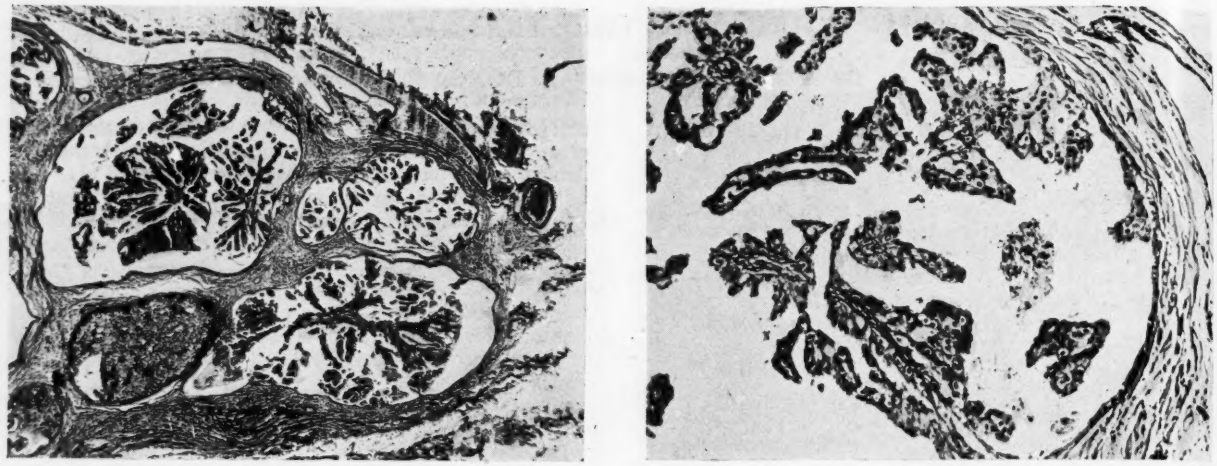
On Examination.—Beneath the areola were two well-defined nodular swellings. They were bluish in colour and gentle pressure caused bright-red blood to flow from the nipple.

The left breast and the axillæ were normal. There were no cutaneous papillomata elsewhere.

At Operation (January, 1944).—The nipple, areola, and surrounding skin containing the nodules were excised.

Convalescence was uneventful.

Microscopical Pathological Examination (Dr. C. V. Harrison).—Section shows extensive intraduct papilloma of the breast (*Fig.* 463).



A B

FIG. 463.—Microphotographs showing extensive intraduct papilloma. A, × 15; B, × 97·5.

COMMENTARY

Duct papilloma is not a common condition, even in the female. Wakeley (1945) has reported a series of 52 cases, but all were in females. I am unable to trace a case of duct papilloma in the male.

The family history of male breast disease is interesting. The age of the patient falls within the commonest age incidence in the female.

Treatment by simple excision is adequate.

REFERENCES

WAKELEY, CECIL P. G. (1945), *Brit. med J.*, **I**, 436.
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A CASE OF CARCINOMA COLI IN A CHILD

BY F. D. SANER

IN the case here reported the previous history, radiographic appearance, and operative findings were typical of an annular stenosing growth of the colon. The unusual feature, of course, is the age, and I am unable to find any detailed record of a similar case.

In Dargeon's book *Cancer in Childhood*, Ewing, referring to the distribution of 183 primary malignant tumours in childhood, mentions 2 as occurring in the rectum and 1 in the stomach; in a further group of 424 childhood cases (Piest) 7 are classified under "Intestine" without any particulars. Both these croups of cases were published some sixty or more years ago. More recently 750 cases of tumours occurring in children under 15 were tabulated at the Mayo Clinic and another group of 215 (infancy to 15 years) at the Memorial Hospital in New York; no mention is made of colonic growths in either of these groups.

I do not wish to convey the impression that a confident diagnosis was made previous to the operation; it was not, but at the same time no satisfactory alternative diagnosis was suggested.

CASE REPORT

HISTORY.—Elizabeth G., aged 11 yr. 1 mth. According to the parents, the child (weight 8 lb. at birth), though always rather tall for her age and thin, had been a healthy child and apart from what are generally referred to as childish complaints, e.g., chicken-pox, etc., had been exceptionally free from minor ailments affecting the gastro-intestinal tract or elsewhere. Nor, so far as the parents knew, was there any history of cancer in either of their own families.

In the middle of May, 1945, the girl had a sharp attack of diarrhœa and vomiting lasting some 24–36 hours, but since the mother had a similar attack at the same time it was attributed to some food irritant. On the last day in May there was a recurrence of abdominal pain and nausea—this time the mother was not affected—which persisted for some 3 or 4 days, when medical advice was sought. At this time it was stated that the bowel was acting, no vomiting was reported, nor did the child appear at all ill. On examination, however, the abdomen was thought to feel "full", though no other signs were detected. The condition remained much the same for a week, after which a definite improvement occurred and was maintained for about a further two weeks, but again the pain returned, was more severe, and was accompanied this time by vomiting and complete arrest of bowel action; when seen some 24 hours later abdominal distension was obvious.

ON ADMISSION.—Shortly after admission a sedative was given, followed some 3 hours later by a glycerin enema which resulted in an action of the bowel, and from then on a steady improvement was maintained without further recurrence of symptoms, and the distension subsided. A week later an X-ray examination (barium meal follow-through) was made which showed the barium to be arrested at a point in the proximal half of the transverse colon; a few days later a barium enema confirmed the findings of the meal (*Figs.* 464, 465).

FIG. 464.—Radiograph following barium meal, showing barium arrested in proximal half of transverse colon.

FIG. 465.—Radiograph following barium enema, and taken 14 days after *Fig.* 464, confirming the site of obstruction.

The upper half of the abdominal wall was at all times kept taut so that no mass was found either on ordinary or at the radiological examination; when the child was anæsthetized it was thought that a small mass could be felt a little above and to the right of the umbilicus, though even then there was considerable doubt.

AT OPERATION.—A laparotomy was performed on July 13 when, at the site shown in the radiographs, a typical annular carcinoma of the transverse colon was found (*Fig.* 466) with obvious involvement of several mesenteric glands close to the bowel wall. No other abnormality was seen. After resection of a length of colon and mesentery the divided ends of the gut were closed and a lateral anastomosis between the proximal and distal segments of the transverse colon was performed.

Recovery from the operation was smooth and uneventful.

THE SPECIMEN.—Dr. P. O. Ellison kindly examined the specimen and has reported as follows: "Segment of transverse colon. The proximal end is ballooned and the distal end contracted; the centre portion is occupied by a neoplasm which completely encircles the bowel; the degree of stenosis is considerable and the lumen of the gut is reduced to a narrow chink. The growth itself, some $\frac{3}{4}$ in. thick, is very firm to touch, with edges everted and surface ulcerated. In the adjacent subperitoneal tissue there are about a dozen scattered nodules varying from 2–3 mm. up to 10 mm. in diameter.

"Microscopical examination reveals a neoplastic process involving the whole bowel wall from mucous membrane to subperitoneal tissue, with the main mass of growth between the muscularis mucosa and muscular coat; there is a considerable degree of fibrosis in this region and the general appearance is not unlike a carcinoma of the stomach.

FIG. 466.—Photograph of portion of colon resected, showing the annular carcinoma.

"The cells are columnar in type, tending to form acini with low columnar and cubical cells which in some

instances are flattened; in general, the cells are solitary or in small groups of the mucus-producing type, and many of them present a signet-ring appearance; in the inner muscular layer there are areas of frank colloidal degeneration. Unlike the usual type of columnar-celled carcinoma, the mucous membrane is not replaced by neoplastic cells; in fact, it is intact and packed with normal mucous glands, though with some indication that the process may have originated in the deepest of these glands.

"Most of the subperitoneal nodules consist of secondary deposits with closely packed acini composed of cells distended with mucus and flattened nuclei producing the signet-ring appearance.

"*Diagnosis*: Stenosing mucoid carcinoma of large intestine."

TRAUMATIC ANEURYSM OF THE COMMON CAROTID ARTERY ASSOCIATED WITH MASSIVE INFARCTION OF THE BRAIN

By NORMAN WHALLEY

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PENETRATING injuries to the neck are always potentially serious problems. On account of their intimate relationships and complete lack of protection, the great vessels, the ninth, tenth, eleventh, and twelfth cranial nerves, the nervous plexuses, and the viscera are all extremely vulnerable to trauma.

Makins (1922), in an analysis of 128 cases of injury to the carotid vessels in the War of 1914–1918, noted that in most cases the wounds of the soft parts were minor in character. He pointed out that no large open wounds of the soft parts were seen in the cases studied and that during a period of three years' duty as Consulting Surgeon he saw no patient in whom the common carotid artery had been tied for control of primary hæmorrhage. The inference is that many carotid injuries die within a short space of time from primary hæmorrhage, and that it is the apparently minor neck injuries which are seen in hospital. In 86 of his 128 cases some form of hæmatoma or traumatic aneurysm developed—a percentage of 67·1. He drew attention to the difficulty caused in diagnosis when cerebral symptoms following an injury to the carotid vessels are complicated by skull fractures, and also drew attention to the fact that injuries to the brachial plexus may cause diagnostic difficulty when occurring in association with cerebral complications.

The case reported below is considered worthy of record as it illustrates the diagnostic difficulties which can occur when cerebral symptoms and neurological signs are found in patients who have suffered both a head injury and a penetrating neck injury.

CASE REPORT

W. T., a German prisoner of war, was wounded in the neck in Normandy on July 8, 1944. When first examined in England on July 9, his records show that he was conscious and co-operative. He had a gunshot wound of entry on the right side of his neck and a gunshot wound of exit on the left side of his neck. The notes stated that there was paralysis of the left arm and hand. Both legs were moving freely on this date. He also had a superficial wound over the right shoulder. There was no further information given. On July 12 he was evacuated to the Newcastle General Hospital under the care of Mr. G. F. Rowbotham. On admission he was drowsy and complained of severe headache.

The following points were noted:—

1. Bruising of the right temporal region of the scalp.
2. Gunshot wound of entry in the right side of the neck overlying the sternomastoid muscle—just below the inferior border of the thyroid cartilage. There was a great deal of bruising and extravasation into the soft tissues.
3. Gunshot wound of exit in the left side of the neck 1 in. above the clavicle near the posterior border of the left sternomastoid muscle.

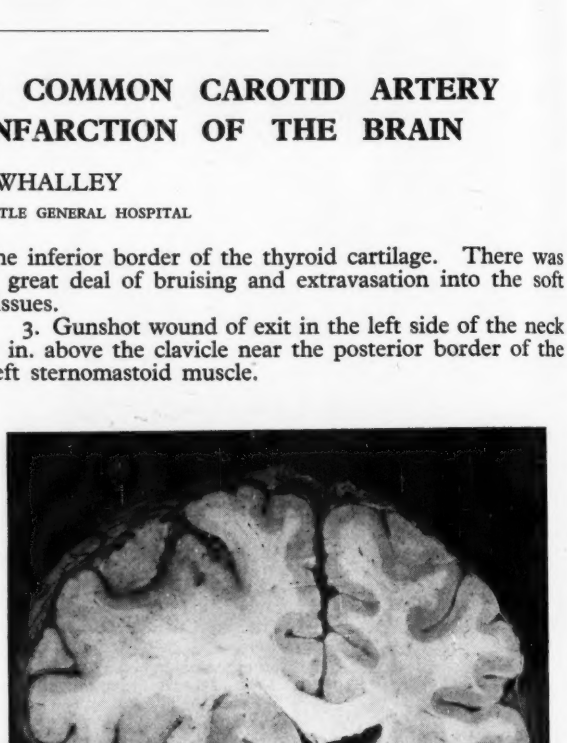

FIG. 467.—Illustrating: (*a*) Traumatic aneurysm of common carotid with thrombosis of the vessels; and (*b*) Infarction of cerebrum and compression of lateral ventricle.

4. Small superficial gunshot wound over the point of the right shoulder.
5. He had a complete hemiplegia on the left side, with absence of the deep tendon reflexes.
6. There were no abnormal neurological signs to indicate injury to any of the cranial nerves.
7. There was no clinical evidence of injury to the sympathetic trunks on either side of the neck.
8. There was no papilloedema.

9. There was diffuse pulsation in the lower part of the neck, and the superficial temporal pulse on the right side was absent.

Radiographs of the skull, neck, chest, and right shoulder showed no evidence of fracture nor the presence of any radio-opaque foreign body.

The diagnosis made by Mr. G. F. Rowbotham was that of traumatic aneurysm of the right common carotid artery, but in view of the bruising in the right temporal region it was considered advisable to explore the right extradural and subdural spaces to exclude, in particular, a subdural hæmatoma. Three days previously the left arm paralysis could well appear to have been due to a brachial plexus injury, particularly in view of the site of the exit wound and the otherwise perfect condition of the patient.

AT OPERATION.—Under local anæsthesia two burr holes were made—one in the right temporal region and the other in the right parietal region. There was no extradural bleeding. The dura mater was tense and pale in colour. On opening the dura mater at both burr holes there was no subdural collection. The brain was very tight and œdematous and tended to protrude through the dural openings.

This minor exploration confirmed the diagnosis and the patient's condition gradually deteriorated and he died on July 14.

POST-MORTEM EXAMINATION.—The missile had penetrated the right sternomastoid muscle and had then caused a large hæmatoma in and around the right carotid sheath. The upper pole of the thyroid gland had been penetrated. The missile had passed behind the œsophagus in a downward direction and to the left, emerging on the left side in front of the scalenus anticus muscle, and had then passed through the posterior part of the left sternomastoid muscle about 1 in. above the clavicle. The brain, the right carotid sheath and contents, the larynx, trachea, œsophagus, thyroid gland, the left common carotid artery, internal carotid artery, external carotid artery, and left brachial plexus (which appeared uninjured) were dissected out and forwarded to Professor A. F. Bernard Shaw, who made a detailed pathological examination and reported as follows :—

Right Carotid Sheath.—This is 8·0 cm. long and the site of diffuse hæmorrhage on its anterior and external surfaces. The common carotid artery from its lower end upwards for 3·0 cm. is intact and the lumen empty ; at this level on its anterior surface, is an aneurysm 0·5 cm. in diameter (*Fig.* 467). A greyish-pink thrombus which fills the sac extends into the remainder of the common carotid and passes upwards to fill the external and internal carotids, each of which is 2·0 cm. long.

Histologically the aneurysm is of traumatic origin, as the wall of the artery has been torn open for a length of 0·8 cm., the gap being filled with thrombus which continues into the carotid sheath ; the latter forms the wall of the sac and none of the components of the arterial wall enter into its composition. No foreign body was found.

The microscopic structure of the thrombus in the three arteries indicates that it has been initiated in the aneurysm and rapidly grown by propagation into the carotids.

The internal jugular vein is empty ; its intima is intact, but at the level of the aneurysm there is subintimal hæmorrhage over a length of 2·0 cm. on the side next to the common carotid.

Left Common, Internal, and External Carotids.—These measure 9.0 cm. The carotid sheath and internal jugular vein are missing. There is slight diffuse hæmorrhage in the adventitia of the carotid vessels, but no other abnormality.

Larynx, Œsophagus, and Thyroid.—Extensive hæmorrhage is present in the fascial plane posterior to the œsophagus and extending over the surface of the right lobe of the thyroid, which is the site of laceration at the upper pole. The œsophagus is intact and there is no hæmorrhage in the tissues between it and the larynx. There is submucosal hæmorrhage in right pyriform fossa, but no trauma. At the posterior commissure of the vocal cords are two small decubitus ulcers.

Left Brachial Plexus.—Shows no trauma or hæmorrhage, but special stains reveal some nerve-fibres with swelling and disintegration of the myelin sheaths.

Brain.—The right internal carotid is filled with thrombus which extends into the right middle and anterior cerebral arteries, occluding the lumina. There is early infarction—confirmed histologically—of the whole of the right cerebral hemisphere, producing intense œdema with compression of the lateral ventricle (*Fig.* 467).

Comment.—The series of events appears to be as follows : Traumatic aneurysm of right common carotid ; hæmorrhage into the tissues of the neck ; thrombosis in the aneurysm with propagation into the right common, external, and internal carotids, right middle and anterior cerebrals, followed by infarction of the right cerebral hemisphere.

The projectile responsible for the trauma was probably of small dimensions, as indicated by the small size of the tears in the common carotid and upper pole of the thyroid gland.

SUMMARY AND DISCUSSION

This patient received a penetrating injury to his neck on July 8, 1944, and when seen on July 9 had a paralysis of the left arm and hand. There are no further clinical records available, but as he was considered fit for evacuation on that date no undue anxiety could have been felt for him. By July 12 he was drowsy, suffered severe headache, and had developed a complete left-sided hemiplegia. Because of the associated head injury it was considered wise to explore the right side of his head to exclude any intracranial hæmorrhage. This negative exploration confirmed the original clinical impression. It would seem that the initial monoplegia was due to an embolus which, originating at the site of injury to the right common carotid artery, lodged in a branch of the middle cerebral artery. Later, progressive thrombosis of the common carotid artery led to complete obstruction and spreading thrombosis in an upward direction, blocking the external and internal carotid arteries, and then, by further extension, the middle and anterior cerebral arteries. The accompanying photograph of the cut brain illustrates extremely well the intense œdema and swelling of the right cerebral hemisphere, secondary to thrombosis of the right middle and anterior cerebral arteries. This thrombosis is a spreading one from the common carotid artery and originates in a traumatic aneurysm. The photograph also shows the post-mortem specimen of the common carotid artery and its two branches with the traumatic aneurysm in situ.

I wish to express my gratitude to Mr. G. F. Rowbotham, Neuro-surgeon, Newcastle-upon-Tyne General Hospital, for his permission to publish this case and for his help and advice. I also desire to thank Professor A. F. Bernard Shaw, of the University of Durham, for the detailed pathological report, the photograph, and for the interest he has shown in this case.

REFERENCE

MAKINS, G. H. (1922), *Official History of the War, Medical Services, Surgery of the War*, **2**, 223.

REVIEWS AND NOTICES OF BOOKS

Illustrations of Regional Anatomy. By E. B. JAMIESON, M.D. Sixth edition. In one bound volume. Pp. xii + 320. 1946. Edinburgh: E. & S. Livingstone Ltd. 75s.

JAMIESON'S *Illustrations* are well known the world over, and now that the seven separate sections have been published in one bound volume surgeons and anatomists alike will benefit in that they have a book of reference. This new sixth edition contains a few new illustrations and an index has been added, which is a real help as any perticular structure may appear in several plates. An excellent volume which will always be popular.

Atlas of Surgical Approaches to Bones and Joints. By TOUFICK NICOLA, M.D., F.A.C.S., Professor of Orthopedics, New York Polyclinic Medical School and Hospital. Pp. 218. 1945. New York: The MacMillan Co. $5.00.

THIS Atlas contains a variety of clear diagrams showing the surgical approach to bones and joints. Major-General Kirk, the Surgeon-General of the United States Army, has written a Foreword in which he states that approximately 70 per cent of war casualties requiring surgical care are primarily extremity injuries. Therefore this book has come at an opportune time, because it reveals to surgeons, both at home and abroad, the surgical approaches to the skeleton.

The book makes clever use of lucid illustrations in conjunction with concise enumeration of each step of action.

To the practising surgeon this pictorial atlas will be invaluable as a quick reference to his current problem. The illustration are clear and the amount of text is reduced to the minimum. The author quite rightly estimates that each illustration is worth 10,000 words of text.

A Textbook of Surgery. By JOHN HOMANS, M.D., Clinical Professor of Surgery, Emeritus. Compiled from Lectures and Other Writings of Members of the Surgical Department of the Harvard Medical School. Sixth edition. 9⅝ × 6⅝ in. Pp. 1278 + viii, with 530 illustrations. 1945. Springfield, Ill.: Charles C. Thomas. (London: Baillière, Tindall & Cox.) $8.00.

THE first edition of this well-known text-book appeared in 1931 and since that date six new editions have been forthcoming; this in itself indicates in no small way the popularity the book enjoys. The new sixth edition shows signs of careful revision and can be said to be right up to date. The use of the sulpha drugs and penicillin in surgery has been given its rightful place. The numerous illustrations are for the most part pen-and-ink drawings, and they have been carefully selected to bring out important points in the text. These illustrations are very refreshing and somewhat novel compared with the usual run of pictures in text-books. We feel sure that this pictorial method of gaining knowledge is of real value to the reader, be he undergraduate or post-graduate.

The chapter dealing with fractures is most comprehensive and practical, and the reader obtains a first-class knowledge of this important branch of surgery. The influence of the second world war has caused some alteration in the treatment of fractures, but on the whole this is very little. All the special branches of surgery are incorporated in this one volume, and Professor Homans has had the good fortune to obtain the services of his surgical colleagues in writing some of the special chapters.

A bibliographical index is incorporated at the end of the book; this is quite a unique feature in a text-book of surgery, but it does serve a useful purpose.

A comprehensive subject index completes the volume.

This text-book will still remain popular as it gives the student just what he requires to know for his final examination in surgery.

Plaster of Paris Technique in the Treatment of Fractures and Other Injuries. By Lt.-Col. T. B. Quigley, *M.C.*, M.D. 9¼ × 6 in. Pp. 107 + xiv, with 103 illustrations. 1945. New York: The Macmillan Company. 18s. net.

THIS volume devoted to the plaster-of-Paris technique is an excellent contribution on the application of this principle. Simplicity is claimed as its keynote, but to this might well be added clearness, for the author by his text and illustrations has made it easy for the reader to understand exactly how and why each step is taken in the application of plaster casts.

The application of plaster to injured limbs in the hands of the expert is simple, and it is therefore satisfactory in this volume to read of the dangers which may be encountered. His advice: "when in doubt, pad a limb before the application of a plaster cast", is excellent. The unpadded plaster, whilst useful and safe over a limited field, is a source of danger in the hands of a novice. The warning of danger which the symptom of pain gives, is above all of importance, and here it is rightly drawn attention to.

Extensile Exposure applied to Limb Surgery. By ARNOLD K. HENRY, M.B. (Dubl.), M.Ch. (Hon., Cairo), F.R.C.S.I., Emeritus Professor of Clinical Surgery in the University of Egypt. 9¼ × 6¾. Pp. 180 + viii, with 127 illustrations. 1945. Edinburgh: E. & S. Livingstone. 30s. net.

THIS book is so much more than a guide to exposure of the long bones that one feels inclined to criticize its title, though it would be difficult to improve upon the author's choice of the term "extensile" to indicate an exposure which, like the tongue of the chameleon, can be made "to reach where it requires". Everyone who has had experience of the surgery of peripheral nerves and blood-vessels knows how often he feels the need for more room, and therefore many besides orthopædic specialists will appreciate the sound practical advice with which this book abounds. Exposure may be extensile not only in length, but in depth and even in conception, amounting almost to the combination of two operations in one. A good example is the exposure of the main neurovascular bundle of the arm through the posterior incision for the musculospiral nerve and humeral shaft. We would suggest that the next edition should show how to display the whole course of the deep femoral in addition to the superficial femoral artery, a problem which taxes the ingenuity of the surgeon who has to hunt for an arteriovenous fistula in the thigh.

But the prime virtue of the book is the stress it lays upon anatomy of the living body in planning the approach to the deepest parts of a limb so that only the minimum of damage may be done to any of its tissues. To do this efficiently the surgeon must be able not only to see but also to feel his way into tissue planes, and Professor Henry is in the good company of the great anatomist-surgeons of the past in advocating the judicious use of

the fingers gently to feel, grasp, or retract muscle bellies or neurovascular bundles. Even taking into consideration all the advantages of the no-touch technique, it must be acknowledged that an instrument can rarely be as gentle and can never be as sensitive as the finger, and with proper precautions the fingers may be used with safety.

The excellence of the illustrations, which must be the product of much study of operations, dissections, and pictures, is beyond all praise, and the whole is flavoured by the author's happy knack of making the English language as extensile as his exposures. It would be well if this book were to be studied by teachers of anatomy as well as by surgeons, since Henry's enthusiasm might even lead to a revival of interest in topographical anatomy which is now so much despised and is therefore so neglected.

Le Catgut : Les Ligatures et les Sutures chirurgicales à travers les Âges. By Dr. A. FANDRE. With a Preface by Professor LOUIS BRUNTZ, Nancy. 9½ × 6¼ in. Pp. 651 + viii, with 137 illustrations. 1944. Paris : Masson et Cie. No price stated.

WHO would have thought that the subject of Catgut deserved a book of 651 pages? Yet this work is interesting and a mine of information, at once historical, scientific, and technical. The first part deals with the early history of the use of ligatures for hæmostasis and quotes from Egyptian, Grecian, and medieval sources. The second part makes a detailed scientific and technical study of catgut, and the third portion deals with the various laws in different countries affecting the preparation of catgut. There is a lengthy bibliography.

This is a good book to keep for reference in a medical library.

A Textbook of Surgery. By American authors. Edited by FREDERICK CHRISTOPHER, B.S., M.D., F.A.C.S., Associate Professor of Surgery, Northwestern University Medical School ; Chief Surgeon, Evanston (Illinois) Hospital. Fourth edition, revised and reset. 9¾ × 6½ in. Pp. 1548 + xxvi, with 1483 illustrations on 764 figures. 1945. London and Philadelphia : W. B. Saunders Co., Ltd. 50s. net.

THE fourth edition of this well-known American textbook of surgery has been completely revised and largely reset. Several new sections have appeared dealing with military surgery, chemotherapy in surgical conditions, and the use of penicillin in surgery.

In a large comprehensive text-book of surgery written by over two hundred contributors it is pleasing to find that the whole range of surgery has been adequately covered. Of necessity some sections are better than others, but Professor Christopher is to be congratulated on the able way he has made use of his contributors so that nothing of importance is missing.

The outstanding sections of the book are by : Adson, dealing with sciatica and cervical rib ; Donald Balfour, writing on tumours of the stomach ; and Elliott Cutler on gastric ulceration.

These are masterful and fascinating to read and are illustrated with original drawings.

There are nearly fifteen hundred illustrations in the book, and on the whole they are of a high order of merit and do enhance the value of the book.

Quite a novel feature of the volume is a complete list of references at the end of each section ; this thoughtful inclusion will prove of real help to many surgeons who want to look up any particular condition more fully. The references are almost entirely American, but this is only to be expected in a text-book written by American authors.

British surgeons will find this volume of value as a book of reference.

An Introduction to Clinical Surgery. Surgical Wherefores and Therefores : A Reasoned Explanation of Surgical Note-taking. By CHARLES F. M. SAINT, C.B.E., M.D., M.S., F.R.C.S. (Eng.), F.R.A.C.S., Professor of Surgery, University of Cape Town. 8½ × 5½ in. Pp. 293 + viii. 1945. Cape Town : Published for the Post-Graduate Press by *The African Bookman*. 25s.

THE appearance of this book is well-timed, for it is essentially clinical in its outlook and is a reasoned explanation for careful surgical case taking. The student of to-day is so often embarrassed by the magnitude of the curriculum that clinical subjects are inclined to be rushed and note taking is scanty or non-existent.

There is also a tendency to-day for diagnosis to be made on various tests, X rays, or blood-counts, to the exclusion of a careful case history and clinical examination.

It is therefore refreshing to read Professor Saint's *Surgical Wherefores and Therefores* ; his mellowed clinical experience has been utilized to the full in this book.

Both the under-graduate and post-graduate will benefit by reading this book, as it contains a vast amount of clinical observations that are not to be found in the average modern text-book.

Adequate treatment cannot be given to any surgical condition unless the diagnosis is correct, and the practitioner will find this book a valuable guide to the diagnostic problems of surgery.

A most useful book and one that surgeons will enjoy reading.

Radiological Examination of the Small Intestine. By ROSS GOLDEN, M.D., Professor of Radiology, College of Physicians and Surgeons, Columbia University ; Director of the Radiological Service, The Presbyterian Hospital, New York. 10 × 7 in. Pp. 239 + xiv, with 75 illustrations. 1945. London : J. B. Lippincott Co. 36s. net.

WITH the increasing use of X rays in abdominal conditions new facts are forthcoming at an ever-increasing rate. The extended use of the Miller-Abbott tube is proving of value not only to the surgeon but also to the radiologist, for its use enables him to portray more exact films showing definite abnormal conditions.

This monograph is the work of many years' careful study, and the author has presented the profession with a useful book of reference in which such conditions as regional enteritis, inflammatory lesions, tuberculous lesions, and neoplasms are beautifully portrayed in X-ray films.

It is pleasing for the reader to be able to see not only the X-ray picture, but also the actual pathological lesion after removal from the patient.

An excellent bibliography is to be found at the end of the book.

El Codo Sus Fracturas y Luxaciones. By D. ANTONIO H.-ROS CODORNIU, Madrid. 9⅝ × 6⅝ in. Pp. 260 + xvi, with 149 illustrations. 1945. Madrid : Cirugia del Aparato Locomotor. Price not stated.

THIS is the second volume of a series of monographs on orthopædics and traumatology. It is perhaps a little prolix, but for the author a labour of love. He covers the ground from comparative anatomy of the joint to a list of salty proverbs relating to its function, such as one would expect Spain to produce.

The mechanics of the complicated movements over the various curvatures of the joint surfaces is studied and

illustrated by good diagrams and pictured models. This is a help to the explanation of the methods of production of the many and diverse injuries to which the joint is subject. The lesions are taken one by one and dealt with as to frequency, pathogenesis, mechanics, diagnosis, progress, treatment, and prognosis.

There is a noticeable preference for operative methods with the use of staples, some of them with close, others very wide, prongs; all of them, of course, of fine calibre. He does not hesitate to use several such staples for multiple injuries of the condyles and shaft, fastening them in place with a wire loop. In the case of the Monteggia injury he figures treatment of the ulna with a long medullary pin introduced through the olecranon. Some forty-six pages are devoted to complicating nerve injuries and their treatment.

The book is well documented with a considerable bibliography and is well produced and copiously illustrated.

Penicillin Therapy and Control in 21 Army Group. Published under the direction of the Director of Medical Services, 21 Army Group, with Introduction by the Consulting Surgeon. 8¼ × 5½ in. Pp. 365. May, 1945.

THIS brochure of some three-hundred-odd pages contains a wealth of information that the average surgeon cannot readily obtain from any other source.

It contains sixty pithy articles dealing with every aspect of wounds due to modern warfare and the treatment of such wounds by penicillin.

Brigadier A. E. Porritt writes the introduction, and one can see his stimulating ideas throughout the book; as consulting surgeon to 21 Army Group he did much to make penicillin available to every wounded man.

It may be said that every aspect of war surgery and medicine, including venereal disease, was treated with penicillin, and the results give the surgeon and physician a very good account of its real value.

BOOK NOTICES

[*The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest to our readers.*]

Anæsthesia in Operations for Goitre. By STANLEY ROWBOTHAM, M.D., D.A., Honorary Anæsthetist to the Royal Free Hospital, to Charing Cross Hospital, and to the Royal Cancer Hospital. 8½ × 5½ in. Pp. 104 + viii, with 54 illustrations. 1945. Oxford: Blackwell Scientific Publications Ltd. 12s. 6d. net.

The Early Diagnosis of the Acute Abdomen. By ZACHARY COPE, B.A., M.D., M.S. (Lond.), F.R.C.S. (Eng.), Surgeon to St. Mary's Hospital; etc. Ninth edition. 8⅜ × 5⅜ in. Pp. 262 + xv, with 38 illustrations. 1946. London: Geoffrey Cumberlege, Oxford University Press. 12s. 6d. net.

Modern Anæsthetic Practice. *The Practitioner Handbooks.* Edited by the late Sir HUMPHREY ROLLESTON, Bt., G.C.V.O., K.C.B., M.D., F.R.C.P., and ALAN MONCRIEFF, M.D., F.R.C.P. Second edition. 8½ × 5½ in. Pp. 150 + viii, with 7 illustrations. 1946. Eyre & Spottiswoode (Publishers) Ltd., for *The Practitioner*. 12s. 6d. net.

Hospital Care of the Surgical Patient. By GEORGE CRILE, jun., M.D., Surgeon, Cleveland Clinic, and FRANKLIN L. SHIVELY, jun., M.D., Assistant Surgeon, Cleveland Clinic. With a Foreword by EVARTS A. GRAHAM. Second edition. 8⅛ × 5⅜ in. Pp. 288 and 18, with 25 illustrations. 1946. Springfield, Ill.: C. C. Thomas. (London: Baillière, Tindal & Cox.) 14s. net.

Fractures of the Jaws. By ROBERT H. IVY, M.D., D.D.S., F.A.C.S., Professor of Plastic Surgery, School of Medicine, University of Pennsylvania; etc.; and LAWRENCE CURTIS, A.B., M.D., D.D.S., F.A.C.S., Associate Professor of Plastic Surgery, School of Medicine, University of Pennsylvania. Third edition, thoroughly revised. 9 × 5¾ in. Pp. 174, with 220 illustrations. 1946. London: Henry Kimpton. 22s. 6d. net.

Choc traumatique. Étude clinique, physio-pathologique et thérapeutique. By J. CREYSSEL, Professeur Agrégé à la Faculté de Lyon; and P. SUIRE, Ex-Chef de Clinique Chirurgicale à la Faculté de Paris. With a Preface by Professor R. LERICHE. 10 × 6½ in. Pp. 310. 1944. Paris: Masson et Cie. Fr. 160.

The 1945 Year Book of Industrial and Orthopedic Surgery. Edited by CHARLES F. PAINTER, M.D., Orthopedic Surgeon to the Massachusetts Women's Hospital and Beth Israel Hospital, Boston. 7 × 4⅝ in. Pp. 432, with 225 illustrations. 1946. Chicago: The Year Book Publishers Inc. (London: H. K. Lewis & Co. Ltd.) 18s. net.

Anatomical Atlas of Orthopædic Operations. By L. S. MICHAELIS, M.D., Orthopædic Surgeon, E.M.S.; Member of the Orthopædic Unit, Botleys Park War Hospital. 9¾ × 7⅜ in. Pp. 67, with 73 illustrations in colour. 1946. London: William Heinemann (Medical Books) Ltd. 25s. net.

A Manual of Surgical Anatomy. By TOM JONES and W. C. SHEPARD. Pocket edition. 7¾ × 5¼ in. Pp. 254 + xvi, with 267 illustrations, 153 in colour. 1945. Philadelphia and London: W. B. Saunders Co. 15s. net.

A Pocket Surgery. By PHILIP H. MITCHINER, C.B., C.B.E., T.D., M.D., M.S. (Lond.), F.R.C.S. (Eng.), D.Ch. Hon. (Durham), D.L., Honorary Surgeon to H.M. The King; Surgeon and Lecturer in Surgery, St. Thomas's Hospital; etc., and A. HEDLEY WHYTE, D.S.O., T.D., M.B., M.S. (Durham), F.R.C.S. (Eng. and Edin.), Honorary Surgeon, Royal Victoria Infirmary, Newcastle-on-Tyne; etc. Second edition. 7¼ × 4¾ in. Pp. 272 + viii. 1946. London: J. & A. Churchill Ltd. 8s. 6d. net.

Hypoglycémies spontanées. Le Traitement chirurgical de l'Hyperinsulinisme. By P. MALLET-GUY, Professeur Agrégé à la Faculté de Médecine de Lyon, and P. MAILLET, Prosecteur à la Faculté de Médecine de Lyon. With a Preface by Professor H. MONDOR. 7¾ × 5⅜ in. Pp. 102, with 17 illustrations. 1944. Paris: Masson et Cie. Fr. 55.

Sciatiques et Lombalgies par Hernie postérieure des Disques intervertébraux. By D. PETIT-DUTAILLIS, Professeur de Pathologie chirurgicale à la Faculté de Médecine; Chirurgien de l'Hôpital Bichat, and S. DE SÈZE, Médecin des Hôpitaux de Paris. 9⅝ × 6⅝ in. Pp. 180, with 133 illustrations. 1945. Paris: Masson et Cie. Fr. 235.

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